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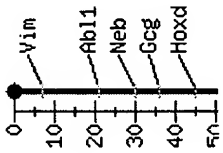
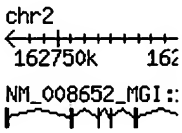
Gene Detail

Symbol	**Mybl2**
Name	**myeloblastosis oncogene-like 2**
ID	MGI:101785
Synonyms	Bmyb
Genetic Map	Chromosome 2 93.0 cM Detailed Genetic Map ± 1 cM Mapping data(2) 
Sequence Map	Chr2:162746128-162776129 bp, + strand (From VEGA annotation of NCBI Build 36) VEGA ContigView | Ensembl ContigView | UCSC Browser | NCBI Map Viewer  MGI Mo
Mammalian homology	human; chimpanzee; dog, domestic; rat (Mammalian Ort Comparative Map (Mouse/Human Mybl2 ± 2 cM) Protein SuperFamily: Myb transforming protein TreeFam: TF326257
Sequences	Representative Sequences Leng ☐ genomic OTTMUSG00000001166 VEGA Gene Model | MGI Sequence Detail 300 ☐ transcript NM_008652 RefSeq | MGI Sequence Detail 37 ☐ polypeptide P48972 UniProt | EBI | MGI Sequence Detail 7 *For the selected sequences* download in FASTA format G All sequences(18)
Phenotypes	All phenotypic alleles(5) : Targeted, knock-out(2) Targeted (1) Mice homozygous for disruptions in this gene die as e after implantation.
Polymorphisms	All PCR and RFLP(2) : PCR(1) RFLP(1) SNPs within 2kb(131 126)

The Jackson Laboratory

last database update
10/04/2007
MGI 3.54

Gene Ontology (GO) classifications	Process: cellular process, regulation of progression Component: intracellular, nucleus... Function: DNA binding All GO classifications(10)
Expression	Theiler Stage 5,17,21,28 Tissues(34) Images(4) Assay Type: Results(34) Assays(3) RNA in situ: 34 3 GXD literature index(4) cDNA source data(10)
Other database links	Ensembl Gene Model: ENSMUSG00000017861 DoTS: DT.532425 DFCI: TC1574940 NIA Mouse Gene Index: U002779 Entrez Gene: 17865 VEGA Gene Model: OTTMUSG00000001166 Gene Traps: AD0109, AM0837, Ayu21-135, CF0716, Cl
Protein domains	InterPro ID Description IPR001005 SANT, DNA-binding IPR012287 Homeodomain-related IPR014778 Myb, DNA-binding IPR015395 C-myb, C-terminal IPR015495 Myb transcription factor Graphical View of Protein Domain Structure
Molecular reagents	All nucleic(11) cDNA(10) Primer pair(1)
References	(Earliest) J:2269 Lam EW *et al.*, "Characterization and cell expression of mouse B-myb." Oncogene 1992 Sep;7(9):18 (Latest) J:102143 Yoshikawa T *et al.*, "High-throughput scr predominantly expressed in the ICM of mouse blastocysts t hybridization." Gene Expr Patterns 2006 Jan;6(2):213-24 All references(13)
Other accession IDs	MGD-MRK-18593

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NEWS 6  JUL 16  CAplus enhanced with French and German abstracts
NEWS 7  JUL 18  CA/CAplus patent coverage enhanced
NEWS 8  JUL 26  USPATFULL/USPAT2 enhanced with IPC reclassification
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          spectral property data
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NEWS 22 SEP 17  CAplus coverage extended to include traditional medicine
          patents
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enhancements
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          Zentralblatt

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          CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
          AND CURRENT DISCOVER FILE IS DATED 19 SEPTEMBER 2007.

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FULL ESTIMATED COST                   0.06      0.27

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=> s mybl2 or myeloblastosis oncogene-like 2
L1        173 MYBL2 OR MYELOBLASTOSIS ONCOGENE-LIKE 2

=> s l1 and (breast cancer or breast carcino?)
  2 FILES SEARCHED...
L2         37 L1 AND (BREAST CANCER OR BREAST CARCINO?)

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3         31 DUP REM L2 (6 DUPLICATES REMOVED)

=> d bib abs 1-
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L3  ANSWER 1 OF 31  CAPLUS  COPYRIGHT 2007 ACS on STN
AN  2007:591350  CAPLUS <<LOGINID::20071005>>
DN  147:28926
TI  Intrinsic genes associated with neoplasms and their use in diagnosis,
    classification and prognosis
IN  Ellis, Matthew; Perou, Charles M.; Ernard, Philip
PA  University of Utah Research Foundation, USA
SO  PCT Int. Appl., 275pp.
    CODEN: PIXXD2
DT  Patent
LA  English
FAN.CNT 1
    PATENT NO.         KIND  DATE        APPLICATION NO.      DATE
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PI  WO 2007061876      A2    20070531    WO 2006-US44737      20061117
    WO 2007061876      A3    20070920
        W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
           CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
           GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN,
           KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK,
           MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO,
           RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT,
           TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
        RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
           IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
           CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,
           GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
           KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA
PRAI US 2005-739155P   P     20051123
AB  A panel of genes that can be used to diagnose and classify neoplasm is
    identified.  These genes are expressed consistently across many cases of
    the same neoplasm and are thus described as "intrinsic" to the neoplasm.
    The panel of genes includes several subsets that can be used to identify
    specific cancers.  The intrinsic genes of  ***breast***  ***cancer***
    show distinct clusterings that give new information about the biol. of
     ***breast***  ***cancer*** .

L3  ANSWER 2 OF 31  CAPLUS  COPYRIGHT 2007 ACS on STN
AN  2007:61688  CAPLUS <<LOGINID::20071005>>
DN  146:135512
TI  A set of 86 genes and methods and kits for predicting and monitoring
    responses to cancer therapy in  ***breast***  ***cancer***
IN  Munnes, Marc; Von Minckwitz, Gunther; Rody, Achim; Kam, Thomas
PA  Bayer Healthcare A.-G., Germany
SO  PCT Int. Appl., 116pp.
    CODEN: PIXXD2
DT  Patent
LA  English
FAN.CNT 1
    PATENT NO.         KIND  DATE        APPLICATION NO.      DATE
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PI  WO 2007006408      A2    20070118    WO 2006-EP6114       20060624
    WO 2007006408      A3    20070823
        W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
           CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
           GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP,
           KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN,
           MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU,
           SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG,
           US, UZ, VC, VN, ZA, ZM, ZW
        RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
           IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
           CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,
           GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
           KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA
PRAI EP 2005-14891     A     20050708
AB  A set of 86 genes that show changes in response to chemotherapy in
    treatment of  ***breast***  ***cancer***  is identified for use in
    diagnosis, prognosis, prevention and treatment of malignant neoplasia and
     ***breast***  ***cancer*** .  The invention further relates to genes
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that are differentially expressed in breast tissue of ***breast*** ***cancer*** patients vs. those of normal "healthy" tissue. Differentially expressed genes for the identification of patients which are likely to respond to chemotherapy are also provided.

L3 ANSWER 3 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2007:333006 CAPLUS <<LOGINID::20071005>>
DN 146:354229
TI Antibodies and tissue microarrays for use in cancer diagnosis, classification and therapy
IN Ring, Brian Z.; Ross, Douglas T.; Seitz, Robert S.; Kirby, Tyler O.; Huh, Warner
PA Applied Genomics, Inc., USA; Uab Research Foundation
SO U.S. Pat. Appl. Publ., 88pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2007065888	A1	20070322	US 2006-432604	20060511
WO 2007086915	A2	20070802	WO 2006-US18332	20060511

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI US 2005-680924P P 20050512
AB A collection of antibodies are provided for classifying tumors, identifying new tumor classes and subclasses, correlating tumor class or subclass with therapeutic regimen or outcome, identifying appropriate (new or known) therapies for particular classes or subclasses, and for predicting outcomes based on class or subclass. Microarray anal. identified 730 candidate genes that (a) are differentially expressed across a set of tumor samples in a manner that suggested they distinguish biol. distinct classes of tumors, (b) were members of a gene functional class that has been linked to cellular pathways implicated in tumor prognosis or drug resistance, and (c) were known or thought to display an expression, localization, modification, or activity pattern that correlates with a relevant tumor feature. Rabbit polyclonal affinity-purified antibodies were then raised against 661 of the proteins encoded by the differentially expressed transcripts, and 460 antibodies were selected further anal. 272 Antibodies exhibited a reproducibly robust staining pattern on tissues relevant for this application, and were applied to a tissue array comprised of .apprx.400 independent breast tumor samples. Nine sub-classes of ***breast*** ***cancer*** patients were identified by their consensus pattern of staining with this ***breast*** ***cancer*** classification sample. Ovarian cancer, lung cancer, and colon cancer classification panels are also identified for classifying tumors. Prognostic value of exemplary panels were assessed by generating Kaplan-Meier recurrence curves, Cox proportional hazard anal., and regression tree anal.

L3 ANSWER 4 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2007:607207 CAPLUS <<LOGINID::20071005>>
DN 147:274342
TI Analytical validation of the Oncotype DX genomic diagnostic test for recurrence prognosis and therapeutic response prediction in node-negative, estrogen receptor-positive ***breast*** ***cancer***
AU Cronin, Maureen; Sangli, Chithra; Liu, Mei-Lan; Pho, Mylan; Dutta, Debjani; Nguyen, Anhthu; Jeong, Jennie; Wu, Jenny; Langone, Clark; Watson, Drew
CS Genomic Health, Inc., Redwood City, CA, USA
SO Clinical Chemistry (Washington, DC, United States) (2007), 53(6), 1084-1091
CODEN: CLCHAU; ISSN: 0009-9147
PB American Association for Clinical Chemistry
DT Journal
LA English
AB Background: Oncotype DX is a clin. validated, high-complexity, multianalyte reverse transcription-PCR genomic test that predicts the likelihood of ***breast*** ***cancer*** recurrence in early-stage, node-neg., estrogen receptor-pos. ***breast*** ***cancer*** . The Recurrence Score (RS) provides a more accurate, reproducible measure of ***breast*** ***cancer*** aggressiveness and therapeutic responsiveness than std. measures. Individualized patient management requires strict performance criteria for clin. lab. tests. We therefore investigated the anal. performance of the assay. Methods: Assays used a pooled RNA sample from fixed paraffin-embedded tissues to evaluate the anal. performance of a 21-gene panel with respect to amplification efficiency, precision, linearity, and dynamic range, as well as limits of detection and quantification. Performance variables were estd. from assays carried out with sample dilns. In addn., individual patient samples were used to test the optimized assay for reproducibility and sources of imprecision. Results: Assay results defined acceptable operational performance ranges, including an estd. max. deviation from linearity of <1 cycle threshold (CT) units over a .gtoreq.2000-fold range of RNA concns., with a mean quantification bias of 0.3% and CVs of 3.2%-5.7%. An anal. of study design showed that assay imprecision contributed by instrument, operator, reagent, and day-to-day baseline variation was low, with SDs of <0.5 CT. Conclusion: The anal. and operational performance specifications defined for the Oncotype DX assay allow the reporting of quant. RS values for individual patients with an SD within 2 RS units on a 100-unit scale.
RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 5 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:470156 CAPLUS <<LOGINID::20071005>>
DN 144:466048
TI Predicting response of cancer patients to chemotherapy using gene expression markers
IN Baker, Joffre B.; Bryant, John L.; Paik, Soonmyung; Shak, Steven
PA Genomic Health, Inc., USA; Nsabp Foundation, Inc.
SO PCT Int. Appl., 53 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006052862	A1	20060518	WO 2005-US40238	20051104

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

AU 2005304824	A1	20060518	AU 2005-304824	20051104
CA 2585571	A1	20060518	CA 2005-2585571	20051104
US 2006166230	A1	20060727	US 2005-267769	20051104
EP 1836629	A1	20070926	EP 2005-817266	20051104

R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR

PRAI US 2004-625485P P 20041105
WO 2005-US40238 W 20051104
AB The present invention provides genes and gene sets useful in predicting the response of cancer, e.g., ***breast*** ***cancer*** , patients to chemotherapy. In addn., the invention provides a clin. validated test, predictive of ***breast*** ***cancer*** patient response to chemotherapy, using multigene RNA anal. In particular, the present inventors identified a set of genes: BCL2; SCUBE2; CCNB1; CTSL2; ESR1; MMP11; ***MYBL2*** ; PGR; STK6; BIRC5 and MMP11, GSTM1, CD68; BAG1; GRB7; ERBB2, which are useful in predicting whether a cancer patient, such as a ***breast*** ***cancer*** patient is likely to show a beneficial response to chemotherapy. Some of these genes are predictive individually, while others are used as part of certain gene groups, used as variables in the methods of the present invention.
RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:471895 CAPLUS <<LOGINID::20071005>>
DN 144:466050
TI Molecular markers of ***breast*** ***cancer*** prognosis and prediction of treatment response
IN Baker, Joffre B.; Bryant, John L.; Paik, Soonmyung; Shak, Steven
PA Genomic Health, Inc., USA; Nsabp Foundation, Inc.
SO PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006052731	A2	20060518	WO 2005-US39970	20051104
WO 2006052731	A3	20070419		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

AU 2005304878	A1	20060518	AU 2005-304878	20051104

CA 2585561 A1 20060518 CA 2005-2585561 20051104
US 2006166231 A1 20060727 US 2005-267807 20051104
EP 1815014 A2 20070808 EP 2005-821698 20051104
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU
PRAI US 2004-625442P P 20041105
WO 2005-US39970 W 20051104
AB The present invention relates to quant. mol. markers that can guide clin. decisions in ***breast*** ***cancer*** , such as estrogen receptor (ESR1)-pos., lymph node-neg. ***breast*** ***cancer*** . In particular, the invention concerns certain genes, the varied expression of which indicates the likelihood of recurrence of surgically resected ***breast*** ***cancer*** in patients who are not treated with a therapeutic agent in the adjuvant setting. In addn., the invention concerns the use of quant. measurement of the expression of certain genes, including the ESR1 gene, that measures as a continuous variable, to det. (a) the likelihood of a beneficial response to the anti-estrogen therapeutic agent, such as tamoxifen; and (b) the potential magnitude of beneficial response to chemotherapy. ***Breast*** ***cancer*** tissue derived from placebo-treated patients was quant. analyzed, using a RT-PCR assay to quantify the expression of sixteen cancer-related genes and five ref. genes. The quant. gene expression anal. resulted in the identification of mol. markers of prognosis. Based on anal. of the relationship between gene expression and distant recurrence-free survival in the placebo arm of the NSABP B-14 trial, a set of genes has been identified, the expression levels of which are indicative of outcome if no further treatment is provided to the patient. Outcome may be manifest in various measurements including survival, recurrence-free survival and distant recurrence-free survival, all of which are within the scope of the invention. The prognostic genes and gene groups identified may be used singly or in particular combinations to predict outcome likelihood for particular patients. Prognostic markers include, specifically, the proliferation group (BIRC5 + MKI + ***MYBL2*** + CCNB1 + STK6), the invasion group (CTSL2 + MMP11), and one or more of the individual genes: CCNB1, BIRC5, ***MYBL2*** , PGR, STK6, MKI, GSTM1, GAPD, RPLPO, and MMP11.

L3 ANSWER 7 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:469903 CAPLUS <<LOGINID::20071005>>
DN 144:486425
TI Alleles of the p53 gene and their effects on gene expression in the classification, prognosis, and diagnosis of cancers
IN Miller, Lance D.; George, Joshy; Vega, Vinsensius B.
PA Agency for Science, Technology and Research, Singapore
SO PCT Int. Appl., 86 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006052218	A1	20060518	WO 2005-SG338	20051005
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
EP 1809762	A1	20070725	EP 2005-788522	20051005
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
IN 2007DN03233	A	20070831	IN 2007-DN3233	20070430

PRAI US 2004-960414 A 20041006
WO 2005-SG338 W 20051005
AB The present invention provides methods, systems and compns. for predicting disease susceptibility in a patient based on correlating alleles of the p53 gene with gene expression profiles for a set of predetd. genes. In some embodiments, methods for the classification, prognosis, and diagnosis of cancers are provided. In other embodiments, the present invention provides statistical methods for building a gene expression-based classifier that may be employed for predicting disease susceptibility in a patient, for classifying carcinomas, and for the prognosis of clin. outcomes. A set of 32 highly informative genes that have their levels of expression by alleles of the p53 gene are described.
RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 8 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:318956 CAPLUS <<LOGINID::20071005>>
DN 144:367977
TI Gene expression profiling in the diagnosis of hematological malignancies
IN Yoganathan, Thillainathan
PA Med Biogene Inc., Can.
SO PCT Int. Appl., 356 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006034573	A1	20060406	WO 2005-CA1464	20050927
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
EP 1805197	A1	20070711	EP 2005-791256	20050927
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				

PRAI US 2004-613980P P 20040927
WO 2005-CA1464 W 20050927
AB Genes showing changes in levels of expression in a no. of hematol. cancers are identified for use in the diagnosis of a broad range of leukemias and lymphomas. Specific sets of genes are identified as diagnostic for specific neoplasms and highly informative subsets of these genes can be used for rapid diagnosis by microarray or soln. hybridization. The combination of probes and the arrays can be used for disease prognosis, diagnosis, staging or grading, treatment management, monitoring of disease progression, predicting disease outcome or complications, and the like. The system of the present invention can be used to profile hematol. cancers selected from the group of lymphoma and leukemia.
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 9 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:75250 CAPLUS <<LOGINID::20071005>>
DN 144:165238
TI Methods for detecting expression levels of housekeeping genes in cancer tissues
IN Szabo, Aniko; Perou, Charles M.; Bernard, Phillip
PA University of Utah Research Foundation, USA; The University of North Carolina at Chapel Hill
SO PCT Int. Appl., 127 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006010150	A2	20060126	WO 2005-US25105	20050715
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
CA 2574447	A1	20060126	CA 2005-2574447	20050715

PRAI US 2004-588222P P 20040715
WO 2005-US25105 W 20050715
AB The invention describes the housekeeping genes expressed in cancer tissues and the methods of detecting and classifying cancer. The expression levels of these genes were detected by real-time RT-PCR. The cancers in human include colon cancer, colon cancer and melanoma. Gene sequences used for anal. were also provided.

L3 ANSWER 10 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:272822 CAPLUS <<LOGINID::20071005>>
DN 144:310064
TI Marker genes for diagnosis of metastatic or primary ***breast*** ***cancer*** and DNA microarrays and diagnostic kits using them
IN Chabaud, Valerie; D' Incan, Chantal; Sylvain, Vidal Valerie; Vidal, Veronique; Agier, Marie; Bignon, Yves Jean; Pradeyrol, Christian
PA Diagnogene S.A, Fr.
SO Fr. Demande, 136 pp.
CODEN: FRXXBL
DT Patent
LA French
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI FR 2875512	A1	20060324	FR 2004-9862	20040917
WO 2006032769	A1	20060330	WO 2005-FR2308	20050919
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,				

CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
PRAI FR 2004-9862 A 20040917
AB Genes showing altered levels of expression in primary and metastatic ***breast*** ***cancer*** are identified and incorporated into microarrays for use in the diagnosis of the disease. DNA microarrays that can be used to rapidly det. the levels of expression of these genes are described for use in diagnostic kits.
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 11 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:195893 CAPLUS <<LOGINID::20071005>>
DN 144:252180
TI DNA microarrays for determination of the sensitivity of a tumor to an anticancer drug and its use in selection of chemotherapies
IN Chabaud, Valerie; D'Incan, Chantal; Sylvain, Vidal Valerie; Vidal, Veronique; Agier, Marie; Bignon, Yves Jean; Pradeyrol, Christian
PA Diagnogene S.A, Fr.
SO Fr. Demande, 109 pp.
CODEN: FRXXBL
DT Patent
LA French
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI FR 2874622	A1	20060303	FR 2004-9192	20040830
WO 2006027463	A2	20060316	WO 2005-FR2142	20050825
WO 2006027463	A3	20060511		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
PRAI FR 2004-9192 A 20040830
AB Genes that show changes in levels of expression in tumors in response to chemotherapeutic agents are identified. The levels of expression of these genes can be used to predict the sensitivity or resistance of the tumor to antitumor agents. The microarray can be used in a test kit to select chemotherapies.
RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 12 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1
AN 2006:785703 CAPLUS <<LOGINID::20071005>>
DN 145:435840
TI Prognosis and Gene Expression Profiling of 20q13-Amplified Breast Cancers
AU Ginestier, Christophe; Cervera, Nathalie; Finetti, Pascal; Esteyries, Severine; Esterni, Benjamin; Adelaide, Jose; Xerri, Luc; Viens, Patrice; Jacquemier, Jocelyne; Charafe-Jauffret, Emmanuelle; Chaffanet, Max; Birnbaum, Daniel; Bertucci, Francois
CS Laboratoire d'Oncologie Moleculaire, Centre de Recherche en Cancerologie de Marseille UMR599 Inserm, Marseille, Fr.
SO Clinical Cancer Research (2006), 12(15), 4533-4544
CODEN: CCREF4; ISSN: 1078-0432
PB American Association for Cancer Research
DT Journal
LA English
AB Purpose: Amplification of chromosomal region 20q13 occurs in ***breast*** ***cancer*** but remains poorly characterized. Exptl. Design: To establish the frequency of 20q13 amplification and select the amplified cases to be studied, we used fluorescence in situ hybridization of bacterial artificial chromosome probes for three 20q13 loci (***MYBL2*** , STK6, ZNF217) on sections of tissue microarrays contg. 466 primary carcinoma samples. We used Affymetryx whole-genome DNA microarrays to establish the gene expression profiles of 20q13-amplified tumors and quant. reverse transcription-PCR to validate the results. Results: We found 36 (8%) 20q13-amplified samples. They were distributed in two types: type 1 tumors showed ZNF217 amplification only, whereas type 2 tumors showed amplification at two or three loci. Examn. of the histoclin. features of the amplified tumors showed two strikingly opposite data. First, type 1 tumors were more frequently lymph node-neg. tumors but were paradoxically assocd. with a poor prognosis. Second, type 2 tumors were more frequently lymph node-pos. tumors but were paradoxically assocd. with a good prognosis. Type 1 and type 2 showed different gene expression profiles. No 20q13 gene could be assocd. with type 1 amplification, whereas several 20q13 genes were overexpressed in type 2 tumors. Conclusions: Our results suggest that amplified tumors of types 1 and 2 are two distinct entities resulting from two different mechanisms and assocd. to different prognosis.
RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 13 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2007:252257 BIOSIS <<LOGINID::20071005>>
DN PREV200700261515
TI The impact on the recurrence score due to patient variation in the quantitative expression of individual genes or gene groups.
AU Watson, D.; Palmer, G.; Baker, J.; Shak, S.
SO Breast Cancer Research and Treatment, (2006) Vol. 100, No. Suppl. 1, pp. S269.
Meeting Info.: 29th Annual San Antonio Breast Cancer Symposium. San Antonio, TX, USA. December 14 -17, 2006. San Antonio Canc Inst; Baylor Coll Med; Canc Therapy & Res Ctr; Univ Texas, Hlth Sci Ctr.
CODEN: BCTRD6. ISSN: 0167-6806.
DT Conference; (Meeting)
Conference; (Meeting Poster)
LA English
ED Entered STN: 25 Apr 2007
Last Updated on STN: 25 Apr 2007

L3 ANSWER 14 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2
AN 2006:1151420 CAPLUS <<LOGINID::20071005>>
DN 146:59911
TI Frequency, prognostic impact, and subtype association of 8p12, 8q24, 11q13, 12p13, 17q12, and 20q13 amplifications in breast cancers
AU Letessier, Anne; Sircoulomb, Fabrice; Ginestier, Christophe; Cervera, Nathalie; Monville, Florence; Gelsi-Boyer, Veronique; Esterni, Benjamin; Geneix, Jeannine; Finetti, Pascal; Zemmour, Christophe; Viens, Patrice; Charafe-Jauffret, Emmanuelle; Jacquemier, Jocelyne; Birnbaum, Daniel; Chaffanet, Max
CS Centre de Recherche en Cancerologie de Marseille, Departement d'Oncologie Moleculaire, UMR599 Inserm/Institut Paoli-Calmettes, Marseille, Fr.
SO BMC Cancer (2006), 6, No pp. given
CODEN: BCMACL; ISSN: 1471-2407
URL: http://www.biomedcentral.com/content/pdf/1471-2407-6-245.pdf
PB BioMed Central Ltd.
DT Journal; (online computer file)
LA English
AB Oncogene amplification and overexpression occur in tumor cells. Amplification status may provide diagnostic and prognostic information and may lead to new treatment strategies. Chromosomal regions 8p12, 8q24, 11q13, 17q12 and 20q13 are recurrently amplified in breast cancers. To assess the frequencies and clin. impact of amplifications, we analyzed 547 invasive breast tumors organized in a tissue microarray (TMA) by fluorescence in situ hybridization (FISH) and calcd. correlations with histoclin. features and prognosis. BAC probes were designed for: (i) two 8p12 subregions centered on RAB11FIP1 and FGFR1 loci, resp.; (ii) 11q13 region centered on CCND1; (iii) 12p13 region spanning NOL1; and (iv) three 20q13 subregions centered on ***MYBL2*** , ZNF217 and AURKA, resp. Regions 8q24 and 17q12 were analyzed with MYC and ERBB2 com. probes, resp.
We obsd. amplification of 8p12 (amplified at RAB11FIP1 and/or FGFR1) in 22.8%, 8q24 in 6.1%, 11q13 in 19.6%, 12p13 in 4.1%, 17q12 in 9.9%, 20q13z (amplified at ZNF217 only) in 9.9%, and 20q13Co (co-amplification of two or three 20q13 loci) in 8.5% of cases. The 8q24, 12p13, and 17q12 amplifications were correlated with high grade. The most frequent single amplifications were 8p12 (9.8%), 8q24 (3.3%) and 12p13 (3.3%), 20q13z and 20q13Co (1.6%) regions. The 17q12 and 11q13 regions were never found amplified alone. The most frequent co-amplification was 8p12/11q13. Amplifications of 8p12 and 17q12 were assocd. with poor outcome. Amplification of 12p13 was assocd. with basal mol. subtype. The results establish the frequencies, prognostic impacts and subtype assocns. of various amplifications and co-amplifications in breast cancers.
RE.CNT 76 THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 15 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:764139 CAPLUS <<LOGINID::20071005>>
DN 145:393796
TI A population-based study of tumor gene expression and risk of ***breast*** ***cancer*** death among lymph node-negative patients
AU Habel, Laurel A.; Shak, Steven; Jacobs, Marlena K.; Capra, Angela; Alexander, Claire; Pho, Mylan; Baker, Joffre; Walker, Michael; Watson, Drew; Hackett, James; Blick, Noelle T.; Greenberg, Deborah; Fehrenbacher, Louis; Langholz, Bryan; Quesenberry, Charles P.
CS Division of Research, Kaiser Permanente, Oakland, CA, USA
SO Breast Cancer Research (2006), 8(3), No pp. given
CODEN: BRCRFS; ISSN: 1465-542X
URL: http://breast-cancer-research.com/content/pdf/bcr1412.pdf
PB BioMed Central Ltd.
DT Journal; (online computer file)

LA English
AB Introduction The Oncotype DX assay was recently reported to predict risk for distant recurrence among a clin. trial population of tamoxifen-treated patients with lymph node-neg., estrogen receptor (ER)-pos. ***breast*** ***cancer*** . To confirm and extend these findings, we evaluated the performance of this 21-gene assay among node-neg. patients from a community hospital setting. Methods A case-control study was conducted among 4,964 Kaiser Permanente patients diagnosed with node-neg. invasive ***breast*** ***cancer*** from 1985 to 1994 and not treated with adjuvant chemotherapy. Cases (n = 220) were patients who died from ***breast*** ***cancer*** . Controls (n = 570) were ***breast*** ***cancer*** patients who were individually matched to cases with respect to age, race, adjuvant tamoxifen, medical facility and diagnosis year, and were alive at the date of death of their matched case. Using an RT-PCR assay, archived tumor tissues were analyzed for expression levels of 16 cancer-related and five ref. genes, and a summary risk score (the Recurrence Score) was calcd. for each patient. Conditional logistic regression methods were used to est. the assocn. between risk of ***breast*** ***cancer*** death and Recurrence Score. Results After adjusting for tumor size and grade, the Recurrence Score was assocd. with risk of ***breast*** ***cancer*** death in ER-pos., tamoxifen-treated and -untreated patients (P = 0.003 and P = 0.03, resp.). At 10 years, the risks for ***breast*** ***cancer*** death in ER-pos., tamoxifen-treated patients were 2.8% (95% confidence interval [CI] 1.7-3.9%), 10.7% (95% CI 6.3-14.9%), and 15.5% (95% CI 7.6-22.8%) for those in the low, intermediate and high risk Recurrence Score groups, resp. They were 6.2% (95% CI 4.5-7.9%), 17.8% (95% CI 11.8-23.3%), and 19.9% (95% CI 14.2-25.2%) for ER-pos. patients not treated with tamoxifen. In both the tamoxifen-treated and -untreated groups, approx. 50% of patients had low risk Recurrence Score values. Conclusion In this large, population-based study of lymph node-neg. patients not treated with chemotherapy, the Recurrence Score was strongly assocd. with risk of ***breast*** ***cancer*** death among ER-pos., tamoxifen-treated and -untreated patients.
RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 16 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:1316274 CAPLUS <<LOGINID::20071005>>
DN 146:119323
TI Gene expression patterns associated with p53 status in ***breast*** ***cancer***
AU Troester, Melissa A.; Herschkowitz, Jason I.; Oh, Daniel S.; He, Xiaping; Hoadley, Katherine A.; Barbier, Claire S.; Perou, Charles M.
CS Division of Biostatistics and Epidemiology, School of Public Health and Health Sciences, University of Massachusetts Amherst, Amherst, MA, USA
SO BMC Cancer (2006), 6, No pp. given
CODEN: BCMACL; ISSN: 1471-2407
URL: http://www.biomedcentral.com/content/pdf/1471-2407-6-276.pdf
PB BioMed Central Ltd.
DT Journal; (online computer file)
LA English
AB Background: ***Breast*** ***cancer*** subtypes identified in genomic studies have different underlying genetic defects. Mutations in the tumor suppressor p53 occur more frequently in estrogen receptor (ER) neg., basal-like and HER2-amplified tumors than in luminal, ER pos. tumors. Thus, because p53 mutation status is tightly linked to other characteristics of prognostic importance, it is difficult to identify p53's independent prognostic effects. The relation between p53 status and subtype can be better studied by combining data from primary tumors with data from isogenic cell line pairs (with and without p53 function). Methods: The p53-dependent gene expression signatures of four cell lines (MCF-7, ZR-75-1, and two immortalized human mammary epithelial cell lines) were identified by comparing p53-RNAi transduced cell lines to their parent cell lines. Cell lines were treated with vehicle only or doxorubicin to identify p53 responses in both non-induced and induced states. The cell line signatures were compared with p53-mutation assocd. genes in breast tumors. Results: Each cell line displayed distinct patterns of p53-dependent gene expression, but cell type specific (basal vs. luminal) commonalities were evident. Further, a common gene expression signature assocd. with p53 loss across all four cell lines was identified. This signature showed overlap with the signature of p53 loss/mutation status in primary breast tumors. Moreover, the common cell-line tumor signature excluded genes that were ***breast*** ***cancer*** subtype-assocd., but not downstream of p53. To validate the biol. relevance of the common signature, we demonstrated that this gene set predicted relapse-free, disease-specific, and overall survival in independent test data. Conclusions: In the presence of ***breast*** ***cancer*** heterogeneity, exptl. and biol.-based methods for assessing gene expression in relation to p53 status provide prognostic and biol.-relevant gene lists. Our biol.-based refinements excluded genes that were assocd. with subtype but not downstream of p53 signaling, and identified a signature for p53 loss that is shared across ***breast*** ***cancer*** subtypes.
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 17 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:1154716 CAPLUS <<LOGINID::20071005>>
DN 143:438041
TI Gene expression markers for predicting response to chemotherapy of cancer
IN Baker, Joffre B.; Shak, Steven; Gianni, Luca
PA Genomic Health, Inc., USA
SO PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005100606	A2	20051027	WO 2005-US11760	20050407
WO 2005100606	A3	20060622		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2005233593	A1	20051027	AU 2005-233593	20050407
CA 2563074	A1	20051027	CA 2005-2563074	20050407
US 2005260646	A1	20051124	US 2005-102228	20050407
EP 1737980	A2	20070103	EP 2005-735478	20050407
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, LV, MK, YU				
KR 2007022694	A	20070227	KR 2006-723413	20061108
PRAI US 2004-561035P	P	20040409		
WO 2005-US11760	W	20050407		

AB Sets of genes that show altered expression in ***breast*** ***cancer*** and that are informative markers for predicting the outcome of chemotherapy are described. Genes that can be used as indicators of successful and unsuccessful outcomes of chemotherapy are described.

L3 ANSWER 18 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:76363 CAPLUS <<LOGINID::20071005>>
DN 142:149724
TI A method of classifying a tumor according to the likelihood of cancer recurrence or response to therapy using expression profile algorithm
IN Baker, Joffre; Bryant, John L.; Paik, Soonmyung; Shak, Steven
PA Genomic Health, Inc., USA
SO PCT Int. Appl., 63 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005008213	A2	20050127	WO 2004-US21163	20040630
WO 2005008213	A3	20050324		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004258085	A1	20050127	AU 2004-258085	20040630
CA 2531967	A1	20050127	CA 2004-2531967	20040630
US 2005048542	A1	20050303	US 2004-883303	20040630
EP 1644858	A2	20060412	EP 2004-777383	20040630
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
JP 2007527220	T	20070927	JP 2006-518761	20040630
PRAI US 2003-486302P	P	20030710		
US 2003-526947P	P	20031203		
WO 2004-US21163	W	20040630		

AB The present invention provides a noninvasive, quant. test for prognosis detn. in cancer patients. The test relies on measurements of the tumor levels of certain mRNAs, particularly, GRB7, HER2, ER, PR, Bcl2, CEGP1, SURV, Ki.67, ***MYBL2*** , CCNB1, STK15, CTSL2, and STMY3. These mRNA levels are inserted into a polynomial formula (algorithm) that yields a numerical recurrence score, which indicates recurrence risk.

L3 ANSWER 19 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:325597 CAPLUS <<LOGINID::20071005>>
DN 142:390446
TI Prediction of likelihood of cancer recurrence by detection of prognostic RNA transcripts in hybridization microarray
IN Baker, Joffre B.; Bryant, John L.; Paik, Soonmyung; Shak, Steven
PA Genomic Health, Inc., USA; Nsabp Foundation, Inc.
SO U.S. Pat. Appl. Publ., 58 pp.
CODEN: USXXCO
DT Patent

LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2005079518	A1	20050414	US 2004-872063	20040617
US 7056674	B2	20060606		
AU 2004283068	A1	20050506	AU 2004-283068	20040617
CA 2530738	A1	20050506	CA 2004-2530738	20040617
WO 2005039382	A2	20050506	WO 2004-US19567	20040617
WO 2005039382	A3	20060112		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1641810	A2	20060405	EP 2004-809450	20040617

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR

JP 2007521005	T	20070802	JP 2006-517415	20040617
US 2006286565	A1	20061221	US 2006-345611	20060131
PRAI US 2003-482339P	P	20030624		
US 2004-872063	A1	20040617		
WO 2004-US19567	W	20040617		

AB The invention relates to a method of predicting the likelihood of long-term survival of a cancer patient by detg. the expression level of one or more prognostic RNA transcripts or their expression products in cancer cells. The present invention provides gene sets the expression of which is important in the diagnosis and/or prognosis of cancer, in particular of ***breast*** ***cancer*** . The expression of one or more of BUB1, C20_orf1, CCNB1, CCNE2, CDC20, CDH1, CTSL2, EPCAM, FOXM1, GRB7, HER2, HNRPAB, Ki-67, KNSL2, LMNB1, MCM2, MELK, MMP12, MMP9, ***MYBL2*** , NEK2, NME1, PCNA, PREP, PTTG1, Src, STK15, STMY3, SURV, TFRC, TOP2A, and TS indicates a decreased likelihood of long-term survival without cancer recurrence. The expression of one or more of BAG1, .beta.-catenin, BIN1, CEGP1, CIAP1; cMYC, DKFZp586M07, DR5, EstR1, GSTM1, GSTM3, ID1, IGF1R, ITGA7, NPD009, PR, and RPLPO indicates an increased likelihood of long-term survival without cancer recurrence. PCR primer/probe set for diagnosis is also provided.

RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 20 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 2007:479066 BIOSIS <<LOGINID::20071005>>
DN PREV200700479166
TI Prediction of response to chemo- and endocrine-therapy.
AU Gianni, L. [Reprint Author]; Pusztai, L.; Baker, J.; Zambetti, M.; Shak, S.
CS Ist Nazl Tumori, I-20133 Milan, Italy
SO EJC Supplements, (OCT 2005) Vol. 3, No. 2, Suppl. S, pp. 31-32. Meeting Info.: 13th European Cancer Conference (ECCO 13). Paris, FRANCE. October 30 -November 03, 2005. ISSN: 1359-6349.
DT Conference; (Meeting) Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 12 Sep 2007 Last Updated on STN: 12 Sep 2007

L3 ANSWER 21 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 2004:177208 BIOSIS <<LOGINID::20071005>>
DN PREV200400179193
TI Tumor tissue microarrays for rapid molecular profiling.
AU Kononen, Juha [Inventor, Reprint Author]; Leighton, Stephen B. [Inventor]; Kallioniemi, Olli-P. [Inventor]
CS Rockville, MD, USA
ASSIGNEE: The United States of America as represented by the Department of Health and Human Services
PI US 6699710 20040302
SO Official Gazette of the United States Patent and Trademark Office Patents, (Mar 2 2004) Vol. 1280, No. 1. http://www.uspto.gov/web/menu/patdata.html. e-file. ISSN: 0098-1133 (ISSN print).
DT Patent
LA English
ED Entered STN: 31 Mar 2004 Last Updated on STN: 31 Mar 2004
AB An array-based technology facilitates rapid correlated gene copy number and expression profiling of very large numbers of human tumors. Hundreds of cylindrical tissue biopsies (diameter 0.6 mm) from morphologically representative regions of individual tumors can be arrayed in a single paraffin block. Consecutive sections from such arrays provide targets for parallel in situ visualization and quantitation of DNA, RNA or protein targets. For example, amplifications of six loci (***mybL2*** , erbB2, Cyclin-D1, myc, 17q23 and 20q13) were rapidly determined by fluorescence in situ hybridization from 372 ethanol-fixed breast cancers. Stratification of tumors by estrogen receptor and p53 expression data revealed distinct patterns of gene amplification in the various subgroups of ***breast*** ***cancer*** that may have prognostic utility. The tissue array technology is useful in the rapid molecular profiling of hundreds of normal and pathological tissue specimens or cultured cells.

L3 ANSWER 22 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:634060 CAPLUS <<LOGINID::20071005>>
DN 141:169028
TI Gene expression markers for ***breast*** ***cancer*** prognosis
IN Cobleigh, Melody A.; Shak, Steve; Baker, Joffre B.; Cronin, Maureen T.
PA Genomic Health, Inc., USA; Rush University Medical Center
SO PCT Int. Appl., 125 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004065583	A2	20040805	WO 2004-US985	20040114
WO 2004065583	A3	20050303		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI

AU 2004205878	A1	20040805	AU 2004-205878	20040114
CA 2513117	A1	20040805	CA 2004-2513117	20040114
US 2004209290	A1	20041021	US 2004-758307	20040114
EP 1587957	A2	20051026	EP 2004-702177	20040114

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

JP 2006516897	T	20060713	JP 2006-500964	20040114
PRAI US 2003-440861P	P	20030115		
WO 2004-US985	W	20040114		

AB The present invention provides a sets of genes, the expression of which is important in the diagnosis and/or prognosis of ***breast*** ***cancer*** . The invention further concerns a prognostic method comprising: subjecting a sample of ***breast*** ***cancer*** cells to quant. anal. of the expression of RNA transcript of atleast one gene selected from the group consisting of GRB7, CD68, CTSL, Chk1, AIB1, CCNB1, MCM2, FBXO5, Her2, STK15, EGFR, ***MYBL2*** , HIF1.alpha. and TS, or their product.

L3 ANSWER 23 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:371064 CAPLUS <<LOGINID::20071005>>
DN 140:373461
TI Evaluation of breast cancer states and outcomes using gene expression profiles
IN West, Mike; Nevins, Joseph R.; Huang, Andrew
PA Synpac, Inc., USA; Duke University
SO PCT Int. Appl., 799 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 5

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004037996	A2	20040506	WO 2003-US33656	20031024
WO 2004037996	A3	20041229		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2004083084	A1	20040429	US 2002-291878	20021112
WO 2004044839	A2	20040527	WO 2002-US38216	20021112

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2004106113	A1	20040603	US 2002-291886	20021112
AU 2003284880	A1	20040513	AU 2003-284880	20031024
PRAI US 2002-420729P	P	20021024		
US 2002-421062P	P	20021025		
US 2002-421102P	P	20021025		

US 2002-424701P P 20021108
US 2002-424715P P 20021108
US 2002-424718P P 20021108
US 2002-291878 A 20021112
US 2002-291886 A 20021112
US 2002-425256P P 20021112
WO 2002-US38216 A 20021112
WO 2002-US38222 A 20021112
US 2003-448461P P 20030221
US 2003-448462P P 20030221
US 2003-457877P P 20030327
US 2003-458373P P 20030331
WO 2003-US33656 W 20031024

AB The present invention relates generally to a method for evaluating and/or predicting breast cancer states and outcomes by measuring gene and metagene expression levels and integrating such data with clin. risk factors. Genes and metagenes whose expressions are correlated with a particular breast cancer risk factor or phenotype are provided using binary prediction tree modeling. The invention provides 175 genes assocd. with metagene predictors of lymph node metastasis, 216 genes assocd. with metagene predictors of breast cancer recurrence, and 496 metagenes related to breast cancer study. Methods of using the subject genes and metagenes in diagnosis and treatment methods, as well as drug screening methods, etc are also provided. In addn., reagents, media and kits that find use in practicing the subject methods are also provided.

L3 ANSWER 24 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN DUPLICATE 3
AN 2004:302197 BIOSIS <<LOGINID::20071005>>
DN PREV200400302676
TI Gene expression analysis in SV40-immortalized human breast luminal epithelial cells with stem cell characteristics using a cDNA microarray.
AU Park, Joon-Suk; Noh, Dong-Young; Kim, Seok-Hyun; Kim, Sung-Hoon; Kong, Gu;
Chang, Chia-Cheng; Lee, Yong-Soon; Trosko, James E.; Kang, Kyung-Sun [Reprint Author]
CS Lab Stem Cell and Tumor BiolDept Vet Publ HlthColl Vet Med, Seoul Natl Univ, San 56-1, Seoul, 151742, South Korea
kangpub@snu.ac.kr
SO International Journal of Oncology, (June 2004) Vol. 24, No. 6, pp. 1545-1558. print.
ISSN: 1019-6439 (ISSN print).
DT Article
LA English
ED Entered STN: 30 Jun 2004
Last Updated on STN: 30 Jun 2004
AB The epithelial compartment of the human breast comprises two distinct cell types. Type I human breast epithelial cells (HBECs) are expressing luminal epithelial cell markers and stem cell characteristics, whereas Type II HBECs show basal epithelial cell phenotypes. When defined in terms of markers for normal cell lineages, most invasive ***breast*** ***cancer*** cells correspond to the phenotype of the common luminal epithelial cell. We had developed simian virus 40-immortalized cell lines from normal HBECs with luminal and stem cell characteristics. To identify molecular changes involved in immortalization, we analyzed the differential gene expression profiles of normal and non-tumorigenic immortalized Type I HBECs using cDNA microarray with 7,448 sequence-verified clones. Out of the 7,448 genes screened, consistent gene expression changes among biological replicates included 67 in Type I HBECs and 86 in Type II HBECs for 4-fold change criteria. Surprisingly, we identified 148 genes (>2.0-fold) as being either up- or down-regulated related to immortalization: 67 genes (***MYBL2*** , UCHL1 et al) were up-regulated, and 81 genes (IGFBP3, CDKN1A et al) were down-regulated significantly. The altered expression levels of the selected genes were subsequently confirmed by semiquantitative RT-PCR. Our studies suggest that the immortalization of Type I HBECs might be an early step in the initiation of a subset of ***breast*** ***cancer*** . Furthermore, these results will open up an avenue for more detailed understanding of breast stem cell and tumor biology.

L3 ANSWER 25 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN
AN 2006:28390 BIOSIS <<LOGINID::20071005>>
DN PREV200600031106
TI Expression of the 21 genes in the Recurrence Score assay and prediction of clinical benefit from tamoxifen in NSABP study B-14 and chemotherapy in NSABP study B-20.
AU Paik, S. [Reprint Author]; Shak, S.; Tang, G.; Kim, C.; Joo, H.; Baker, J.; Cronin, M.; Watson, D.; Braynt, J.; Costantino, J.; Wolmark, N.
CS NSABP, Pittsburgh, PA USA
SO Breast Cancer Research and Treatment, (2004) Vol. 88, No. Suppl. 1, pp. S15.
Meeting Info.: 27th Annual Charles A Coltman San Antonio Breast Cancer Symposium. San Antonio, TX, USA. December 08 -11, 2004.
CODEN: BCTRD6. ISSN: 0167-6806.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 28 Dec 2005
Last Updated on STN: 28 Dec 2005

L3 ANSWER 26 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:754553 CAPLUS <<LOGINID::20071005>>
DN 137:227626
TI Methods for diagnosing and monitoring malignancies by screening gene copy numbers
IN Kuo, Wen-Lin; Polikoff, Daniel; Pinkel, Daniel; Albertson, Donna; Berchuk, Andy; Gray, Joe W.
PA The Regents of the University of California, USA
SO PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002077197	A2	20021003	WO 2002-US9419	20020327
WO 2002077197	A3	20031023		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003077582	A1	20030424	US 2001-819148	20010327
AU 2002303165	A1	20021008	AU 2002-303165	20020327
PRAI US 2001-819148	A	20010327		
WO 2002-US9419	W	20020327		

AB The invention concerns the discovery that an amplification of some genes or an increase in that gene activity and a deletion of some genes or a decrease in that gene activity is a marker for the presence of, progression of, or predisposition to, a cancer (e.g., ***breast*** ***cancer***). Using this information, this invention provides methods of detecting a predisposition to cancer in an animal. The methods involve (i) providing a biol. sample from an animal (e.g. a human patient); (ii) detecting the level of the genes of the present invention within the biol. sample; and (iii) comparing the level of one or more of said genes with a level of one or more of said genes in a control sample taken from a normal, cancer-free tissue.

L3 ANSWER 27 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:200545 CAPLUS <<LOGINID::20071005>>
DN 136:353411
TI Silence of chromosomal amplifications in colon cancer
AU Platzer, Petra; Upender, Madhvi B.; Wilson, Keith; Willis, Joseph; Lutterbaugh, James; Nosrati, Arman; Willson, James K. V.; Mack, David; Ried, Thomas; Markowitz, Sanford
CS Howard Hughes Medical Institute, Cleveland, OH, 44106, USA
SO Cancer Research (2002), 62(4), 1134-1138
CODEN: CNREA8; ISSN: 0008-5472
PB American Association for Cancer Research
DT Journal
LA English
AB Oncogene activation by gene amplification is a major pathogenetic mechanism in human cancer. Using comparative genomic hybridization, we detd. that metastatic human colon cancers commonly acquire numerous extra copies of chromosome arms 7p, 8q, 13q, and 20q. We then examd. the consequence of these amplifications on gene expression using DNA microarrays. Of 55,000 transcripts profiled, 2146 were detd. to map to one of the four common colon cancer amplicons and to also be expressed in normal or malignant colon tissues. Of these, only 81 transcripts (3.8%) demonstrated a 2-fold increase over normal expression among cancers bearing the corresponding chromosomal amplification. Chromosomal amplifications are common in colon cancer metastasis, but increased expression of genes within these amplicons is rare.
RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 28 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN DUPLICATE 4
AN 2000:436918 BIOSIS <<LOGINID::20071005>>
DN PREV200000436918
TI Comparative genomic hybridization analysis of 38 ***breast*** ***cancer*** cell lines: A basis for interpreting complementary DNA microarray data.
AU Forozan, Farahnaz; Mahlamaki, Eija H.; Monni, Outi; Chen, Yidong; Veldman, Robin; Jiang, Yuan; Gooden, Gerald C.; Ethier, Stephen P.; Kallioniemi, Anne; Kallioniemi, Olli-P. [Reprint author]
CS Cancer Genetics Branch, National Human Genome Research Institute, NIH, 49
Convent Drive, Building 49, Room 4A24, Bethesda, MD, 20892-4470, USA
SO Cancer Research, (August 15, 2000) Vol. 60, No. 16, pp. 4519-4525. print.
CODEN: CNREA8. ISSN: 0008-5472.
DT Article
LA English
ED Entered STN: 11 Oct 2000
Last Updated on STN: 10 Jan 2002
AB ***Breast*** ***cancer*** cell lines provide a useful starting

point for the discovery and functional analysis of genes involved in ***breast*** ***cancer*** . Here, we studied 38 established ***breast*** ***cancer*** cell lines by comparative genomic hybridization (CGH) to determine recurrent genetic alterations and the extent to which these cell lines resemble uncultured tumors. The following chromosomal gains were observed: 8q (75%), 1q (61%), 20q (55%), 7p (44%), 3q (39%), 5p (39%), 7q (39%), 17q (33%), 1p (30%), and 20p (30%), and the most common losses were: 8p (58%), 18q (58%), 1p (42%), Xp (42%), Xq (42%), 4p (36%), 11q (36%), 18p (33%), 10q (30%), and 19p (28%). Furthermore, 35 recurrent high-level amplification sites were identified, most often involving 8q23 (37%), 20q13 (29%), 3q25-q26 (24%), 17q22-q23 (16%), 17q23-q24 (16%), 1p13 (11%), 1q32 (11%), 5p13 (11%), 5p14 (11%), 11q13 (11%), 17q12-q21 (11%), and 7q21-q22 (11%). A comparison of DNA copy number changes found in the cell lines with those reported in 17 published studies (698 tumors) of uncultured tumors revealed a substantial degree of overlap. CGH copy number profiles may facilitate identification of important new genes located at the hotspots of such chromosomal alterations. This was illustrated by analyzing expression levels of 1236 genes using cDNA microarrays in four of the cell lines. Several highly overexpressed genes (such as RCH1 at 17q23, TOPO II at 17q21-q22, as well as CAS and ***MYBL2*** at 20q13) were involved in these recurrent DNA amplifications. In conclusion, DNA copy number profiles were generated by CGH for most of the publicly available ***breast*** ***cancer*** cell lines and were made available on a web site (http://www.nhgri.nih.gov/DIR/CGB/CR2000). This should facilitate the correlative analysis of gene expression and copy number as illustrated here by the finding by cDNA microarrays of several overexpressed genes that were amplified.

L3 ANSWER 29 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:3213 CAPLUS <<LOGINID::20071005>>
DN 135:74810
TI Gene expression profiling of primary ***breast*** ***carcinomas*** using arrays of candidate genes
AU Bertucci, Francois; Houlgatte, Remi; Benziane, Athmane; Granjeaud, Samuel; Adelaide, Jose; Tagett, Rebecca; Loriod, Beatrice; Jacquemier, Jocelyne; Viens, Patrice; Jordan, Bertrand; Birnbaum, Daniel; Nguyen, Catherine
CS Laboratoire de Biologie des Tumeurs, TAGC, Institut Paoli-Calmettes (IPC), Marseille, Fr.
SO Human Molecular Genetics (2000), 9(20), 2981-2991
CODEN: HMGEE5; ISSN: 0964-6906
PB Oxford University Press
DT Journal
LA English
AB ***Breast*** ***cancer*** is characterized by an important histoclin. heterogeneity that currently hampers the selection of the most appropriate treatment for each case. This problem could be solved by the identification of new parameters that better predict the natural history of the disease and its sensitivity to treatment. A large-scale mol. characterization of ***breast*** ***cancer*** could help in this context. Using cDNA arrays, we studied the quant. mRNA expression levels of 176 candidate genes in 34 primary ***breast*** ***carcinomas*** along three directions: comparison of tumor samples, correlations of mol. data with conventional histoclin. prognostic features and gene correlations. The study evidenced extensive heterogeneity of breast tumors at the transcriptional level. A hierarchical clustering algorithm identified two molecularly distinct subgroups of tumors characterized by a different clin. outcome after chemotherapy. This outcome could not have been predicted by the commonly used histoclin. parameters. No correlation was found with the age of patients, tumor size, histol. type and grade. However, expression of genes was differential in tumors with lymph node metastasis and according to the estrogen receptor status; ERBB2 expression was strongly correlated with the lymph node status ($P < 0.0001$) and that of GATA3 with the presence of estrogen receptors ($P < 0.001$). Thus, our results identified new ways to group tumors according to outcome and new potential targets of carcinogenesis. They show that the systematic use of cDNA array testing holds great promise to improve the classification of ***breast*** ***cancer*** in terms of prognosis and chemosensitivity and to provide new potential therapeutic targets.
RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 30 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 5
AN 2000:401121 CAPLUS <<LOGINID::20071005>>
DN 133:294517
TI Frequent amplification of chromosomal region 20q12-q13 in ovarian cancer
AU Tanner, Minna M.; Grenman, Seija; Koul, Anjila; Johannsson, Oskar; Meltzer, Paul; Pejovic, Tanja; Borg, Ake; Isola, Jorma J.
CS Laboratory of Cancer Genetics, Tampere University and University Hospital, Tampere, FIN-33101, Finland
SO Clinical Cancer Research (2000), 6(5), 1833-1839
CODEN: CCREF4; ISSN: 1078-0432
PB American Association for Cancer Research
DT Journal
LA English
AB DNA amplification at chromosomal region 20q12-q13, which is common in ***breast*** ***cancer*** , has recently been described also in ovarian tumors. We studied the amplification of the recently identified candidate oncogenes in this region in 24 sporadic, 3 familial and 4 hereditary ovarian carcinomas, and in 8 ovarian cancer cell lines. High-level amplification of at least one of the five nonsyntenic regions at 20q12-q13.2 was found in 13 sporadic (54%) and in all four hereditary tumors. Typically, two or more distinct amplicons (sepd. by nonamplified DNA) were found coamplified in various combinations. The regions defined by the AIB1 and PTPN1 genes (at 20q12 and 20q13.1, resp.) were amplified in 25% and 29% of the sporadic tumors, also without simultaneous coamplification of other regions. Amplification of AIB1 (a steroid receptor coactivator gene) was assocd. with estrogen receptor positivity in sporadic ovarian carcinomas ($P = 0.01$) and showed a tendency to correlate with poor survival of patients. Of the genes amplified in ***breast*** ***cancer*** , the BTAK gene was amplified in 21%, the ***MYBL2*** gene in 17%, and the ZNF217 gene in 12.5% of the sporadic tumors. The high frequency of gene amplification at 20q12-q13.2 suggests that the genes amplified therein may play a central role in the pathogenesis of sporadic and hereditary ovarian carcinoma.
RE.CNT 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 31 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:566265 CAPLUS <<LOGINID::20071005>>
DN 131:181940
TI Tumor tissue microarrays for rapid molecular profiling
IN Leighton, Stephen B.; Kononen, Juha; Kallioniemi, Olli
PA The United States of America, Represented by the Secretary, Department of Health, USA
SO PCT Int. Appl., 36 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9944063	A2	19990902	WO 1999-US4001	19990224
WO 9944063	A3	19991104		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2318984	A1	19990902	CA 1999-2318984	19990224
AU 9928757	A	19990915	AU 1999-28757	19990224
AU 754047	B2	20021031		
EP 1068528	A2	20010117	EP 1999-909585	19990224
EP 1068528	B1	20060802		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
JP 2002505432	T	20020219	JP 2000-533760	19990224
AT 335199	T	20060815	AT 1999-909585	19990224
EP 1715340	A2	20061025	EP 2006-16017	19990224
EP 1715340	A3	20061129		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
ES 2272057	T3	20070416	ES 1999-909585	19990224
US 6699710	B1	20040302	US 2000-622686	20001012
US 2002192702	A1	20021219	US 2002-215762	20020809
US 2003138827	A1	20030724	US 2002-305800	20021126
US 2006166253	A1	20060727	US 2006-374178	20060314
PRAI US 1998-75979P	P	19980225		
EP 1999-909585	A3	19990224		
WO 1999-US4001	W	19990224		
US 2000-622686	A3	20001012		
US 2002-305800	A3	20021126		

AB An array-based technol. facilitates rapid correlated gene copy no. and expression profiling of a very large nos. of human tumors. Hundreds of cylindrical tissue biopsies (diam. 0.6 mm) from morphol. representative regions of individual tumors can be arrayed in a single paraffin block. Consecutive sections from such arrays provide targets for parallel in situ visualization and quantitation of DNA, RNA or protein targets. For example, amplifications of six loci (***mybL2*** , erbB2, Cyclin-D1, myc, 17q23 and 20q13) were rapidly detd. by fluorescence in situ hybridization from 372 ethanol-fixed breast cancers. Stratification of tumors by estrogen receptor and p53 expression data revealed distinct patterns of gene amplification in the various subgroups of ***breast*** ***cancer*** that may have prognostic utility. The tissue array technol. is useful in the rapid mol. profiling of hundreds of normal and pathol. tissue specimens or cultured cells.

=> FIL STNGUIDE

COST IN U.S. DOLLARS	SINCE FILE ENTRY	SESSION	TOTAL
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COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 0.30 113.56

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE TOTAL
ENTRY SESSION
CA SUBSCRIBER PRICE 0.00 -19.50

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=> s breast (cancer or carcin?)
MISSING OPERATOR 'BREAST (CANCER'
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s breast (3a) (cancer or carcin?)
L4 352725 BREAST (3A) (CANCER OR CARCIN?)

=> s l4 and (prognosis or surviv?)
L5 62163 L4 AND (PROGNOSIS OR SURVIV?)

=> s l5 and gene profile
L6 23 L5 AND GENE PROFILE

=> s l6 and review
L7 3 L6 AND REVIEW

=> dup rem l7
PROCESSING COMPLETED FOR L7
L8 3 DUP REM L7 (0 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L8 ANSWER 1 OF 3 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2007256910 EMBASE <<LOGINID::20071005>>
TI Individualization of therapy using mammaprint.RTM..RTM.: From development to the MINDACT trial.
AU Mook S.; Van't Veer L.J.; Rutgers E.J.T.; Piccart-Gebhart M.J.; Cardoso F.
CS Dr. F. Cardoso, Medical Oncology and Translational Research, Jules Bordet Institute, Boulevard de Waterloo, 125, 1000 Brussels, Belgium. fatima.cardoso@bordet.be
SO Cancer Genomics and Proteomics, (May 2007) Vol. 4, No. 3, pp. 147-156.
Refs: 28
ISSN: 1109-6535
CY Greece
DT Journal; General Review; (Review)
FS 016 Cancer
022 Human Genetics
027 Biophysics, Bioengineering and Medical Instrumentation
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
ED Entered STN: 12 Jun 2007
Last Updated on STN: 12 Jun 2007
AB To date, most treatment decisions for adjuvant chemotherapy in ***breast*** ***cancer*** are based on conventional clinicopathological criteria. Since ***breast*** ***cancer*** tumors with similar clinicopathological characteristics can have strikingly different outcomes, the current selection for adjuvant chemotherapy is far from accurate. Using high-throughput microarray analysis, a 70-gene signature was identified which can accurately select early stage ***breast*** ***cancer*** patients who are highly likely to develop distant metastases, and therefore, may benefit the most from adjuvant chemotherapy. This ***review*** describes the development of the 70- ***gene*** ***profile*** (Mammaprint.RTM..RTM.), its retrospective validation and feasibility studies, and its prospective validation in the large adjuvant MINDACT (Microarray In Node-negative Disease may Avoid ChemoTherapy) clinical trial.

L8 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:351455 CAPLUS <<LOGINID::20071005>>
DN 144:429966
TI Prognostic significance of cyclin E in ***breast*** ***cancer***
AU Potemski, Piotr; Kordek, Radzislaw
CS Klinika Chemioterapii Nowotworow, Katedra Onkol., Wojewodzki Szpital Specjalistyczny im. Mikolaja Kopernika, Uniw. Med., Lodz, 93-509, Pol.
SO Onkologia w Praktyce Klinicznej (2005), 1(2), 76-82
CODEN: OPKNBB; ISSN: 1734-3542
PB Wydawnictwo Via Medica
DT Journal; General Review
LA Polish
AB A ***review*** . In the present ***review*** the role of cyclin E and other cell cycle mols. as prognostic markers in ***breast*** ***cancer*** is discussed Cell cycle transitions are controlled by cyclin-dependent protein kinases. Cyclin E is an important regulator of cell cycle progression from phase G1 to synthetic phase. The highest concn. of cyclin E in the cell is obsd. near restriction point in the late G1 phase. In ***breast*** ***cancer*** the aberrant expression of cyclin E has been frequently described. In ***breast*** ***cancer*** cells full-length cyclin E is degradated by proteolysis to its low mol. wt. isoforms. Overexpression of both cyclin E and low mol. wt. isoforms gives tumor cells growth advantage. Expression of cyclin E and other cell cycle mols. can be evaluated by immunohistochem., tissue microarrays, various blot assays, reverse transcription-polymerase chain reaction and DNA microarrays. The results of numerous studies show that overexpression of cyclin E may be an important prognostic factor. However, in some models of multivariate anal. this effect was eliminated by estrogen receptors presence or other classical prognostic factors. However, futher confirmatory studies are warranted as overexpression of cyclin E may also be just a part of ***gene*** ***profile*** and not a single, independent prognostic factor.

L8 ANSWER 3 OF 3 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2001092515 EMBASE <<LOGINID::20071005>>
TI DNA microarrays in pediatric cancer.
AU Triche T.J.; Schofield D.; Buckley J.
CS Dr. T.J. Triche, Children's Hospital, MS43, 4650 Sunset Boulevard, Los Angeles, CA 90027, United States
SO Cancer Journal, (2001) Vol. 7, No. 1, pp. 2-15.
Refs: 68
ISSN: 1528-9117 CODEN: CAJOCB
CY United States
DT Journal; General Review; (Review)
FS 016 Cancer
007 Pediatrics and Pediatric Surgery
LA English
SL English
ED Entered STN: 29 Mar 2001
Last Updated on STN: 29 Mar 2001
AB Childhood cancer, like all cancer, is at heart a genetic disease. Consequently, fundamental understanding of the oncogenic process is likely to be beneficially addressed by genetic methodology. Current methods have largely focused on single-gene defects, like chimeric genes, which are present in many sarcomas and leukemias. Real understanding is more likely to derive from a genome-wide analysis of these malignancies. Recent technologic advances have made it possible to simultaneously assess the entire expressed ***gene*** ***profile*** , or transcriptome, of a given cancer. Foremost among these methods is gene expression profiling using DNA microarrays. Two basic approaches predominate: spotted arrays and photolithography arrays. Regardless of the method, the resulting information can be used to create disease profiles, but only if appropriate bioinformatic solutions are employed. Common analytic approaches include two-way expression comparisons, or scatter analyses; outlier gene analysis, to identify significantly dysregulated genes: dendrogram analyses, as pioneered by Eisen; cluster analyses to identify diagnostic or biologic groups; and various forms of functional analyses to identify relevant genes and biologic pathways. Studies of both adult and pediatric cancer have demonstrated the feasibility of such analyses to identify both diagnostic and prognostic groups of tumors. Acute childhood leukemias have been grouped into myelogenous and lymphoid, and even B- and T-cell subsets. ***Breast*** ***cancer*** prognostic groups have been identified on the basis of a small subset of expressed genes. In addition, preliminary data on childhood sarcomas appear to identify both diagnostic and prognostic subsets. Specifically, embryonal rhabdomyosarcoma could be distinguished from alveolar rhabdomyosarcoma, and even morphologically mixed embryonal and alveolar rhabdomyosarcoma showed similar gene expression profiles in both histologies. Further, collaborative studies using clustering analyses appear to identify prognostic groups of diverse sarcomas. Larger institutional and cooperative group studies are currently underway to validate these preliminary findings.

=> FIL STNGUIDE
COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 31.87 145.43

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ENTRY SESSION
CA SUBSCRIBER PRICE -0.78 -20.28

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=> FIL BIOSIS CAPLUS EMBASE
COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 0.18 145.61

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE TOTAL
ENTRY SESSION
CA SUBSCRIBER PRICE 0.00 -20.28

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=> d his

(FILE 'HOME' ENTERED AT 16:41:09 ON 05 OCT 2007)

FILE 'STNGUIDE' ENTERED AT 16:41:16 ON 05 OCT 2007

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 16:41:21 ON 05 OCT 2007
L1 173 S MYBL2 OR MYELOBLASTOSIS ONCOGENE-LIKE 2
L2 37 S L1 AND (BREAST CANCER OR BREAST CARCINO?)
L3 31 DUP REM L2 (6 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 16:53:01 ON 05 OCT 2007

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 16:55:48 ON 05 OCT 2007
L4 352725 S BREAST (3A) (CANCER OR CARCIN?)
L5 62163 S L4 AND (PROGNOSIS OR SURVIV?)
L6 23 S L5 AND GENE PROFILE
L7 3 S L6 AND REVIEW
L8 3 DUP REM L7 (0 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 16:59:31 ON 05 OCT 2007

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 17:01:28 ON 05 OCT 2007

=> s l5 and mybl2
L9 26 L5 AND MYBL2

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10 23 DUP REM L9 (3 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 23 ANSWERS - CONTINUE? Y/(N):y

L10 ANSWER 1 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2007:591350 CAPLUS <<LOGINID::20071005>>
DN 147:28926
TI Intrinsic genes associated with neoplasms and their use in diagnosis, classification and ***prognosis***
IN Ellis, Matthew; Perou, Charles M.; Ernard, Philip
PA University of Utah Research Foundation, USA
SO PCT Int. Appl., 275pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2007061876	A2	20070531	WO 2006-US44737	20061117
WO 2007061876	A3	20070920		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRAI US 2005-739155P P 20051123
AB A panel of genes that can be used to diagnose and classify neoplasm is identified. These genes are expressed consistently across many cases of the same neoplasm and are thus described as "intrinsic" to the neoplasm. The panel of genes includes several subsets that can be used to identify specific cancers. The intrinsic genes of ***breast*** ***cancer*** show distinct clusterings that give new information about the biol. of ***breast*** ***cancer*** .

L10 ANSWER 2 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2007:61688 CAPLUS <<LOGINID::20071005>>
DN 146:135512
TI A set of 86 genes and methods and kits for predicting and monitoring responses to ***cancer*** therapy in ***breast*** ***cancer***
IN Munnes, Marc; Von Minckwitz, Gunther; Rody, Achim; Karn, Thomas
PA Bayer Healthcare A.-G., Germany
SO PCT Int. Appl., 116pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2007006408	A2	20070118	WO 2006-EP6114	20060624
WO 2007006408	A3	20070823		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRAI EP 2005-14891 A 20050708
AB A set of 86 genes that show changes in response to chemotherapy in treatment of ***breast*** ***cancer*** is identified for use in diagnosis, ***prognosis*** , prevention and treatment of malignant neoplasia and ***breast*** ***cancer*** . The invention further relates to genes that are differentially expressed in ***breast*** tissue of ***breast*** ***cancer*** patients vs. those of normal "healthy" tissue. Differentially expressed genes for the identification of patients which are likely to respond to chemotherapy are also provided.

L10 ANSWER 3 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2007:333006 CAPLUS <<LOGINID::20071005>>
DN 146:354229
TI Antibodies and tissue microarrays for use in cancer diagnosis, classification and therapy
IN Ring, Brian Z.; Ross, Douglas T.; Seitz, Robert S.; Kirby, Tyler O.; Huh, Warner
PA Applied Genomics, Inc., USA; Uab Research Foundation
SO U.S. Pat. Appl. Publ., 88pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2007065888	A1	20070322	US 2006-432604	20060511
WO 2007086915	A2	20070802	WO 2006-US18332	20060511

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI US 2005-680924P P 20050512
AB A collection of antibodies are provided for classifying tumors, identifying new tumor classes and subclasses, correlating tumor class or subclass with therapeutic regimen or outcome, identifying appropriate (new or known) therapies for particular classes or subclasses, and for predicting outcomes based on class or subclass. Microarray anal. identified 730 candidate genes that (a) are differentially expressed across a set of tumor samples in a manner that suggested they distinguish biol. distinct classes of tumors, (b) were members of a gene functional class that has been linked to cellular pathways implicated in tumor ***prognosis*** or drug resistance, and (c) were known or thought to display an expression, localization, modification, or activity pattern that correlates with a relevant tumor feature. Rabbit polyclonal affinity-purified antibodies were then raised against 661 of the proteins encoded by the differentially expressed transcripts, and 460 antibodies were selected further anal. 272 Antibodies exhibited a reproducibly robust staining pattern on tissues relevant for this application, and were applied to a tissue array comprised of .apprx.400 independent breast tumor samples. Nine sub-classes of ***breast*** ***cancer*** patients were identified by their consensus pattern of staining with this ***breast*** ***cancer*** classification sample. Ovarian cancer,

lung cancer, and colon cancer classification panels are also identified for classifying tumors. Prognostic value of exemplary panels were assessed by generating Kaplan-Meier recurrence curves, Cox proportional hazard anal., and regression tree anal.

L10 ANSWER 4 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2007:607207 CAPLUS <<LOGINID::20071005>>
DN 147:274342
TI Analytical validation of the Oncotype DX genomic diagnostic test for recurrence ***prognosis*** and therapeutic response prediction in node-negative, estrogen receptor-positive ***breast*** ***cancer***
AU Cronin, Maureen; Sangli, Chithra; Liu, Mei-Lan; Pho, Mylan; Dutta, Debjani; Nguyen, Anhthu; Jeong, Jennie; Wu, Jenny; Langone, Clark; Watson, Drew
CS Genomic Health, Inc., Redwood City, CA, USA
SO Clinical Chemistry (Washington, DC, United States) (2007), 53(6), 1084-1091
CODEN: CLCHAU; ISSN: 0009-9147
PB American Association for Clinical Chemistry
DT Journal
LA English
AB Background: Oncotype DX is a clin. validated, high-complexity, multianalyte reverse transcription-PCR genomic test that predicts the likelihood of ***breast*** ***cancer*** recurrence in early-stage, node-neg., estrogen receptor-pos. ***breast*** ***cancer*** . The Recurrence Score (RS) provides a more accurate, reproducible measure of ***breast*** ***cancer*** aggressiveness and therapeutic responsiveness than std. measures. Individualized patient management requires strict performance criteria for clin. lab. tests. We therefore investigated the anal. performance of the assay. Methods: Assays used a pooled RNA sample from fixed paraffin-embedded tissues to evaluate the anal. performance of a 21-gene panel with respect to amplification efficiency, precision, linearity, and dynamic range, as well as limits of detection and quantification. Performance variables were estd. from assays carried out with sample dilns. In addn., individual patient samples were used to test the optimized assay for reproducibility and sources of imprecision. Results: Assay results defined acceptable operational performance ranges, including an estd. max. deviation from linearity of <1 cycle threshold (CT) units over a .gtoreq.2000-fold range of RNA concns., with a mean quantification bias of 0.3% and CVs of 3.2%-5.7%. An anal. of study design showed that assay imprecision contributed by instrument, operator, reagent, and day-to-day baseline variation was low, with SDs of <0.5 CT. Conclusion: The anal. and operational performance specifications defined for the Oncotype DX assay allow the reporting of quant. RS values for individual patients with an SD within 2 RS units on a 100-unit scale.
RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 5 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:471895 CAPLUS <<LOGINID::20071005>>
DN 144:466050
TI Molecular markers of ***breast*** ***cancer*** ***prognosis*** and prediction of treatment response
IN Baker, Joffre B.; Bryant, John L.; Paik, Soonmyung; Shak, Steven
PA Genomic Health, Inc., USA; Nsabp Foundation, Inc.
SO PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006052731	A2	20060518	WO 2005-US39970	20051104
WO 2006052731	A3	20070419		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
AU 2005304878	A1	20060518	AU 2005-304878	20051104
CA 2585561	A1	20060518	CA 2005-2585561	20051104
US 2006166231	A1	20060727	US 2005-267807	20051104
EP 1815014	A2	20070808	EP 2005-821698	20051104
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				
PRAI US 2004-625442P	P	20041105		
WO 2005-US39970	W	20051104		

AB The present invention relates to quant. mol. markers that can guide clin. decisions in ***breast*** ***cancer*** , such as estrogen receptor (ESR1)-pos., lymph node-neg. ***breast*** ***cancer*** . In particular, the invention concerns certain genes, the varied expression of which indicates the likelihood of recurrence of surgically resected ***breast*** ***cancer*** in patients who are not treated with a therapeutic agent in the adjuvant setting. In addn., the invention concerns the use of quant. measurement of the expression of certain genes, including the ESR1 gene, that measures as a continuous variable, to det. (a) the likelihood of a beneficial response to the anti-estrogen therapeutic agent, such as tamoxifen; and (b) the potential magnitude of beneficial response to chemotherapy. ***Breast*** ***cancer*** tissue derived from placebo-treated patients was quant. analyzed, using a RT-PCR assay to quantify the expression of sixteen cancer-related genes and five ref. genes. The quant. gene expression anal. resulted in the identification of mol. markers of ***prognosis*** . Based on anal. of the relationship between gene expression and distant recurrence-free ***survival*** in the placebo arm of the NSABP B-14 trial, a set of genes has been identified, the expression levels of which are indicative of outcome if no further treatment is provided to the patient. Outcome may be manifest in various measurements including ***survival*** , recurrence-free ***survival*** and distant recurrence-free ***survival*** , all of which are within the scope of the invention. The prognostic genes and gene groups identified may be used singly or in particular combinations to predict outcome likelihood for particular patients. Prognostic markers include, specifically, the proliferation group (BIRC5 + MKI + ***MYBL2*** + CCNB1 + STK6), the invasion group (CTSL2 + MMP11), and one or more of the individual genes: CCNB1, BIRC5, ***MYBL2*** , PGR, STK6, MKI, GSTM1, GAPD, RPLPO, and MMP11.

L10 ANSWER 6 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:469903 CAPLUS <<LOGINID::20071005>>
DN 144:486425
TI Alleles of the p53 gene and their effects on gene expression in the classification, ***prognosis*** , and diagnosis of cancers
IN Miller, Lance D.; George, Joshy; Vega, Vinsensius B.
PA Agency for Science, Technology and Research, Singapore
SO PCT Int. Appl., 86 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006052218	A1	20060518	WO 2005-SG338	20051005
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
EP 1809762	A1	20070725	EP 2005-788522	20051005
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
IN 2007DN03233	A	20070831	IN 2007-DN3233	20070430
PRAI US 2004-960414	A	20041006		
WO 2005-SG338	W	20051005		

AB The present invention provides methods, systems and compns. for predicting disease susceptibility in a patient based on correlating alleles of the p53 gene with gene expression profiles for a set of predetd. genes. In some embodiments, methods for the classification, ***prognosis*** , and diagnosis of cancers are provided. In other embodiments, the present invention provides statistical methods for building a gene expression-based classifier that may be employed for predicting disease susceptibility in a patient, for classifying carcinomas, and for the ***prognosis*** of clin. outcomes. A set of 32 highly informative genes that have their levels of expression by alleles of the p53 gene are described.
RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 7 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:318956 CAPLUS <<LOGINID::20071005>>
DN 144:367977
TI Gene expression profiling in the diagnosis of hematological malignancies
IN Yoganathan, Thillainathan
PA Med Biogene Inc., Can.
SO PCT Int. Appl., 356 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006034573	A1	20060406	WO 2005-CA1464	20050927
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

EP 1805197 A1 20070711 EP 2005-791256 20050927

R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR

PRAI US 2004-613980P P 20040927

WO 2005-CA1464 W 20050927

AB Genes showing changes in levels of expression in a no. of hematol. cancers are identified for use in the diagnosis of a broad range of leukemias and lymphomas. Specific sets of genes are identified as diagnostic for specific neoplasms and highly informative subsets of these genes can be used for rapid diagnosis by microarray or soln. hybridization. The combination of probes and the arrays can be used for disease ***prognosis*** , diagnosis, staging or grading, treatment management, monitoring of disease progression, predicting disease outcome or complications, and the like. The system of the present invention can be used to profile hematol. cancers selected from the group of lymphoma and leukemia.

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 8 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2006:272822 CAPLUS <<LOGINID::20071005>>

DN 144:310064

TI Marker genes for diagnosis of metastatic or primary breast cancer and DNA microarrays and diagnostic kits using them

IN Chabaud, Valerie; D' Incan, Chantal; Sylvain, Vidal Valerie; Vidal, Veronique; Agier, Marie; Bignon, Yves Jean; Pradeyrol, Christian

PA Diagnogene S.A, Fr.

SO Fr. Demande, 136 pp.

CODEN: FRXXBL

DT Patent

LA French

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI FR 2875512	A1	20060324	FR 2004-9862	20040917
WO 2006032769	A1	20060330	WO 2005-FR2308	20050919

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI FR 2004-9862 A 20040917

AB Genes showing altered levels of expression in primary and metastatic breast cancer are identified and incorporated into microarrays for use in the diagnosis of the disease. DNA microarrays that can be used to rapidly det. the levels of expression of these genes are described for use in diagnostic kits.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 9 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2006:195893 CAPLUS <<LOGINID::20071005>>

DN 144:252180

TI DNA microarrays for determination of the sensitivity of a tumor to an anticancer drug and its use in selection of chemotherapies

IN Chabaud, Valerie; D'Incan, Chantal; Sylvain, Vidal Valerie; Vidal, Veronique; Agier, Marie; Bignon, Yves Jean; Pradeyrol, Christian

PA Diagnogene S.A, Fr.

SO Fr. Demande, 109 pp.

CODEN: FRXXBL

DT Patent

LA French

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI FR 2874622	A1	20060303	FR 2004-9192	20040830
WO 2006027463	A2	20060316	WO 2005-FR2142	20050825
WO 2006027463	A3	20060511		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI FR 2004-9192 A 20040830

AB Genes that show changes in levels of expression in tumors in response to chemotherapeutic agents are identified. The levels of expression of these genes can be used to predict the sensitivity or resistance of the tumor to antitumor agents. The microarray can be used in a test kit to select chemotherapies.

RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 10 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1

AN 2006:785703 CAPLUS <<LOGINID::20071005>>

DN 145:435840

TI ***Prognosis*** and Gene Expression Profiling of 20q13-Amplified Breast Cancers

AU Ginestier, Christophe; Cervera, Nathalie; Finetti, Pascal; Esteyries, Severine; Esterni, Benjamin; Adelaide, Jose; Xerri, Luc; Viens, Patrice; Jacquemier, Jocelyne; Charafe-Jauffret, Emmanuelle; Chaffanet, Max; Birnbaum, Daniel; Bertucci, Francois

CS Laboratoire d'Oncologie Moleculaire, Centre de Recherche en Cancerologie de Marseille UMR599 Inserm, Marseille, Fr.

SO Clinical Cancer Research (2006), 12(15), 4533-4544

CODEN: CCREF4; ISSN: 1078-0432

PB American Association for Cancer Research

DT Journal

LA English

AB Purpose: Amplification of chromosomal region 20q13 occurs in ***breast*** ***cancer*** but remains poorly characterized. Exptl. Design: To establish the frequency of 20q13 amplification and select the amplified cases to be studied, we used fluorescence in situ hybridization of bacterial artificial chromosome probes for three 20q13 loci (***MYBL2*** , STK6, ZNF217) on sections of tissue microarrays contg. 466 primary carcinoma samples. We used Affymetryx whole-genome DNA microarrays to establish the gene expression profiles of 20q13-amplified tumors and quant. reverse transcription-PCR to validate the results. Results: We found 36 (8%) 20q13-amplified samples. They were distributed in two types: type 1 tumors showed ZNF217 amplification only, whereas type 2 tumors showed amplification at two or three loci. Examn. of the histoclin. features of the amplified tumors showed two strikingly opposite data. First, type 1 tumors were more frequently lymph node-neg. tumors but were paradoxically assocd. with a poor ***prognosis*** . Second, type 2 tumors were more frequently lymph node-pos. tumors but were paradoxically assocd. with a good ***prognosis*** . Type 1 and type 2 showed different gene expression profiles. No 20q13 gene could be assocd. with type 1 amplification, whereas several 20q13 genes were overexpressed in type 2 tumors. Conclusions: Our results suggest that amplified tumors of types 1 and 2 are two distinct entities resulting from two different mechanisms and assocd. to different ***prognosis*** .

RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 11 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 2007:252257 BIOSIS <<LOGINID::20071005>>

DN PREV200700261515

TI The impact on the recurrence score due to patient variation in the quantitative expression of individual genes or gene groups.

AU Watson, D.; Palmer, G.; Baker, J.; Shak, S.

SO Breast Cancer Research and Treatment, (2006) Vol. 100, No. Suppl. 1, pp. S269.

Meeting Info.: 29th Annual San Antonio Breast Cancer Symposium. San Antonio, TX, USA. December 14 -17, 2006. San Antonio Canc Inst; Baylor Coll Med; Canc Therapy & Res Ctr; Univ Texas, Hlth Sci Ctr.

CODEN: BCTRD6. ISSN: 0167-6806.

DT Conference; (Meeting)

Conference; (Meeting Poster)

LA English

ED Entered STN: 25 Apr 2007

Last Updated on STN: 25 Apr 2007

L10 ANSWER 12 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2

AN 2006:1151420 CAPLUS <<LOGINID::20071005>>

DN 146:59911

TI Frequency, prognostic impact, and subtype association of 8p12, 8q24, 11q13, 12p13, 17q12, and 20q13 amplifications in breast cancers

AU Letessier, Anne; Sircoulomb, Fabrice; Ginestier, Christophe; Cervera, Nathalie; Monville, Florence; Gelsi-Boyer, Veronique; Esterni, Benjamin; Geneix, Jeannine; Finetti, Pascal; Zemmour, Christophe; Viens, Patrice; Charafe-Jauffret, Emmanuelle; Jacquemier, Jocelyne; Birnbaum, Daniel; Chaffanet, Max

CS Centre de Recherche en Cancerologie de Marseille, Departement d'Oncologie Moleculaire, UMR599 Inserm/Institut Paoli-Calmettes, Marseille, Fr.

SO BMC Cancer (2006), 6, No pp. given

CODEN: BCMACL; ISSN: 1471-2407

URL: http://www.biomedcentral.com/content/pdf/1471-2407-6-245.pdf

PB BioMed Central Ltd.

DT Journal; (online computer file)

LA English

AB Oncogene amplification and overexpression occur in tumor cells. Amplification status may provide diagnostic and prognostic information and may lead to new treatment strategies. Chromosomal regions 8p12, 8q24, 11q13, 17q12 and 20q13 are recurrently amplified in breast cancers. To assess the frequencies and clin. impact of amplifications, we analyzed 547 invasive breast tumors organized in a tissue microarray (TMA) by fluorescence in situ hybridization (FISH) and calcd. correlations with histoclin. features and ***prognosis*** . BAC probes were designed for: (i) two 8p12 subregions centered on RAB11FIP1 and FGFR1 loci, resp.; (ii) 11q13 region centered on CCND1; (iii) 12p13 region spanning NOL1; and (iv) three 20q13 subregions centered on ***MYBL2*** , ZNF217 and AURKA, resp. Regions 8q24 and 17q12 were analyzed with MYC and ERBB2 com. probes, resp. We obsd. amplification of 8p12 (amplified at RAB11FIP1 and/or FGFR1) in 22.8%, 8q24 in 6.1%, 11q13 in 19.6%, 12p13 in 4.1%, 17q12 in 9.9%, 20q13z (amplified at ZNF217 only) in 9.9%, and 20q13Co (co-amplification of two or three 20q13 loci) in 8.5% of cases. The 8q24, 12p13, and 17q12 amplifications were correlated with high grade. The most frequent single amplifications were 8p12 (9.8%), 8q24 (3.3%) and 12p13 (3.3%), 20q13z and 20q13Co (1.6%) regions. The 17q12 and 11q13 regions were never found amplified alone. The most frequent co-amplification was 8p12/11q13. Amplifications of 8p12 and 17q12 were assocd. with poor outcome. Amplification of 12p13 was assocd. with basal mol. subtype. The results establish the frequencies, prognostic impacts and subtype assocns. of various amplifications and co-amplifications in breast cancers.
RE.CNT 76 THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 13 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:764139 CAPLUS <<LOGINID::20071005>>
DN 145:393796
TI A population-based study of tumor gene expression and risk of ***breast*** ***cancer*** death among lymph node-negative patients
AU Habel, Laurel A.; Shak, Steven; Jacobs, Marlena K.; Capra, Angela; Alexander, Claire; Pho, Mylan; Baker, Joffre; Walker, Michael; Watson, Drew; Hackett, James; Blick, Noelle T.; Greenberg, Deborah; Fehrenbacher, Louis; Langholz, Bryan; Quesenberry, Charles P.
CS Division of Research, Kaiser Permanente, Oakland, CA, USA
SO Breast Cancer Research (2006), 8(3), No pp. given
CODEN: BRCRFS; ISSN: 1465-542X
URL: http://breast-cancer-research.com/content/pdf/bcr1412.pdf
PB BioMed Central Ltd.
DT Journal; (online computer file)
LA English
AB Introduction The Oncotype DX assay was recently reported to predict risk for distant recurrence among a clin. trial population of tamoxifen-treated patients with lymph node-neg., estrogen receptor (ER)-pos. ***breast*** ***cancer*** . To confirm and extend these findings, we evaluated the performance of this 21-gene assay among node-neg. patients from a community hospital setting. Methods A case-control study was conducted among 4,964 Kaiser Permanente patients diagnosed with node-neg. invasive ***breast*** ***cancer*** from 1985 to 1994 and not treated with adjuvant chemotherapy. Cases (n = 220) were patients who died from ***breast*** ***cancer*** . Controls (n = 570) were ***breast*** ***cancer*** patients who were individually matched to cases with respect to age, race, adjuvant tamoxifen, medical facility and diagnosis year, and were alive at the date of death of their matched case. Using an RT-PCR assay, archived tumor tissues were analyzed for expression levels of 16 cancer-related and five ref. genes, and a summary risk score (the Recurrence Score) was calcd. for each patient. Conditional logistic regression methods were used to est. the assocn. between risk of ***breast*** ***cancer*** death and Recurrence Score. Results After adjusting for tumor size and grade, the Recurrence Score was assocd. with risk of ***breast*** ***cancer*** death in ER-pos., tamoxifen-treated and -untreated patients (P = 0.003 and P = 0.03, resp.). At 10 years, the risks for ***breast*** ***cancer*** death in ER-pos., tamoxifen-treated patients were 2.8% (95% confidence interval [CI] 1.7-3.9%), 10.7% (95% CI 6.3-14.9%), and 15.5% (95% CI 7.6-22.8%) for those in the low, intermediate and high risk Recurrence Score groups, resp. They were 6.2% (95% CI 4.5-7.9%), 17.8% (95% CI 11.8-23.3%), and 19.9% (95% CI 14.2-25.2%) for ER-pos. patients not treated with tamoxifen. In both the tamoxifen-treated and -untreated groups, approx. 50% of patients had low risk Recurrence Score values. Conclusion In this large, population-based study of lymph node-neg. patients not treated with chemotherapy, the Recurrence Score was strongly assocd. with risk of ***breast*** ***cancer*** death among ER-pos., tamoxifen-treated and -untreated patients.
RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 14 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:1316274 CAPLUS <<LOGINID::20071005>>
DN 146:119323
TI Gene expression patterns associated with p53 status in ***breast*** ***cancer***
AU Troester, Melissa A.; Herschkowitz, Jason I.; Oh, Daniel S.; He, Xiaping; Hoadley, Katherine A.; Barbier, Claire S.; Perou, Charles M.
CS Division of Biostatistics and Epidemiology, School of Public Health and Health Sciences, University of Massachusetts Amherst, Amherst, MA, USA
SO BMC Cancer (2006), 6, No pp. given
CODEN: BCMACL; ISSN: 1471-2407
URL: http://www.biomedcentral.com/content/pdf/1471-2407-6-276.pdf
PB BioMed Central Ltd.
DT Journal; (online computer file)
LA English
AB Background: ***Breast*** ***cancer*** subtypes identified in genomic studies have different underlying genetic defects. Mutations in the tumor suppressor p53 occur more frequently in estrogen receptor (ER) neg., basal-like and HER2-amplified tumors than in luminal, ER pos. tumors. Thus, because p53 mutation status is tightly linked to other characteristics of prognostic importance, it is difficult to identify p53's independent prognostic effects. The relation between p53 status and subtype can be better studied by combining data from primary tumors with data from isogenic cell line pairs (with and without p53 function). Methods: The p53-dependent gene expression signatures of four cell lines (MCF-7, ZR-75-1, and two immortalized human mammary epithelial cell lines) were identified by comparing p53-RNAi transduced cell lines to their parent cell lines. Cell lines were treated with vehicle only or doxorubicin to identify p53 responses in both non-induced and induced states. The cell line signatures were compared with p53-mutation assocd. genes in breast tumors. Results: Each cell line displayed distinct patterns of p53-dependent gene expression, but cell type specific (basal vs. luminal) commonalities were evident. Further, a common gene expression signature assocd. with p53 loss across all four cell lines was identified. This signature showed overlap with the signature of p53 loss/mutation status in primary breast tumors. Moreover, the common cell-line tumor signature excluded genes that were ***breast*** ***cancer*** subtype-assocd., but not downstream of p53. To validate the biol. relevance of the common signature, we demonstrated that this gene set predicted relapse-free, disease-specific, and overall ***survival*** in independent test data. Conclusions: In the presence of ***breast*** ***cancer*** heterogeneity, exptl. and biol.-based methods for assessing gene expression in relation to p53 status provide prognostic and biol.-relevant gene lists. Our biol.-based refinements excluded genes that were assocd. with subtype but not downstream of p53 signaling, and identified a signature for p53 loss that is shared across ***breast*** ***cancer*** subtypes.
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 15 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:1154716 CAPLUS <<LOGINID::20071005>>
DN 143:438041
TI Gene expression markers for predicting response to chemotherapy of cancer
IN Baker, Joffre B.; Shak, Steven; Gianni, Luca
PA Genomic Health, Inc., USA
SO PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005100606	A2	20051027	WO 2005-US11760	20050407
WO 2005100606	A3	20060622		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2005233593	A1	20051027	AU 2005-233593	20050407
CA 2563074	A1	20051027	CA 2005-2563074	20050407
US 2005260646	A1	20051124	US 2005-102228	20050407
EP 1737980	A2	20070103	EP 2005-735478	20050407
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, LV, MK, YU				
KR 2007022694	A	20070227	KR 2006-723413	20061108
PRAI US 2004-561035P	P	20040409		
WO 2005-US11760	W	20050407		

AB Sets of genes that show altered expression in ***breast*** ***cancer*** and that are informative markers for predicting the outcome of chemotherapy are described. Genes that can be used as indicators of successful and unsuccessful outcomes of chemotherapy are described.

L10 ANSWER 16 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:76363 CAPLUS <<LOGINID::20071005>>
DN 142:149724
TI A method of classifying a tumor according to the likelihood of cancer recurrence or response to therapy using expression profile algorithm
IN Baker, Joffre; Bryant, John L.; Paik, Soonmyung; Shak, Steven
PA Genomic Health, Inc., USA
SO PCT Int. Appl., 63 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO. KIND DATE APPLICATION NO. DATE

PI WO 2005008213 A2 20050127 WO 2004-US21163 20040630
WO 2005008213 A3 20050324
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
AU 2004258085 A1 20050127 AU 2004-258085 20040630
CA 2531967 A1 20050127 CA 2004-2531967 20040630
US 2005048542 A1 20050303 US 2004-883303 20040630
EP 1644858 A2 20060412 EP 2004-777383 20040630
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK
JP 2007527220 T 20070927 JP 2006-518761 20040630
PRAI US 2003-486302P P 20030710
US 2003-526947P P 20031203
WO 2004-US21163 W 20040630

AB The present invention provides a noninvasive, quant. test for ***prognosis*** detn. in cancer patients. The test relies on measurements of the tumor levels of certain mRNAs, particularly, GRB7, HER2, ER, PR, Bcl2, CEGP1, SURV, Ki.67, ***MYBL2*** , CCNB1, STK15, CTSL2, and STMY3. These mRNA levels are inserted into a polynomial formula (algorithm) that yields a numerical recurrence score, which indicates recurrence risk.

L10 ANSWER 17 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:325597 CAPLUS <<LOGINID::20071005>>
DN 142:390446
TI Prediction of likelihood of cancer recurrence by detection of prognostic RNA transcripts in hybridization microarray
IN Baker, Joffre B.; Bryant, John L.; Paik, Soonmyung; Shak, Steven
PA Genomic Health, Inc., USA; Nsabp Foundation, Inc.
SO U.S. Pat. Appl. Publ., 58 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO. KIND DATE APPLICATION NO. DATE

PI US 2005079518 A1 20050414 US 2004-872063 20040617
US 7056674 B2 20060606
AU 2004283068 A1 20050506 AU 2004-283068 20040617
CA 2530738 A1 20050506 CA 2004-2530738 20040617
WO 2005039382 A2 20050506 WO 2004-US19567 20040617
WO 2005039382 A3 20060112
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
EP 1641810 A2 20060405 EP 2004-809450 20040617
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR
JP 2007521005 T 20070802 JP 2006-517415 20040617
US 2006286565 A1 20061221 US 2006-345611 20060131
PRAI US 2003-482339P P 20030624
US 2004-872063 A1 20040617
WO 2004-US19567 W 20040617

AB The invention relates to a method of predicting the likelihood of long-term ***survival*** of a cancer patient by detg. the expression level of one or more prognostic RNA transcripts or their expression products in cancer cells. The present invention provides gene sets the expression of which is important in the diagnosis and/or ***prognosis*** of ***cancer*** , in particular of ***breast*** ***cancer*** . The expression of one or more of BUB1, C20_orf1, CCNB1, CCNE2, CDC20, CDH1, CTSL2, EPCAM, FOXM1, GRB7, HER2, HNRPAB, Ki-67, KNSL2, LMNB1, MCM2, MELK, MMP12, MMP9, ***MYBL2*** , NEK2, NME1, PCNA, PREP, PTTG1, Src, STK15, STMY3, SURV, TFRC, TOP2A, and TS indicates a decreased likelihood of long-term ***survival*** without cancer recurrence. The expression of one or more of BAG1, .beta.-catenin, BIN1, CEGP1, ClAP1; cMYC, DKFZp586M07, DR5, EstR1, GSTM1, GSTM3, ID1, IGF1R, ITGA7, NPD009, PR, and RPLPO indicates an increased likelihood of long-term ***survival*** without cancer recurrence. PCR primer/probe set for diagnosis is also provided.

RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 18 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:634060 CAPLUS <<LOGINID::20071005>>
DN 141:169028
TI Gene expression markers for ***breast*** ***cancer*** ***prognosis***
IN Cobleigh, Melody A.; Shak, Steve; Baker, Joffre B.; Cronin, Maureen T.
PA Genomic Health, Inc., USA; Rush University Medical Center
SO PCT Int. Appl., 125 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO. KIND DATE APPLICATION NO. DATE

PI WO 2004065583 A2 20040805 WO 2004-US985 20040114
WO 2004065583 A3 20050303
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI
AU 2004205878 A1 20040805 AU 2004-205878 20040114
CA 2513117 A1 20040805 CA 2004-2513117 20040114
US 2004209290 A1 20041021 US 2004-758307 20040114
EP 1587957 A2 20051026 EP 2004-702177 20040114
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
JP 2006516897 T 20060713 JP 2006-500964 20040114
PRAI US 2003-440861P P 20030115
WO 2004-US985 W 20040114

AB The present invention provides a sets of genes, the expression of which is important in the diagnosis and/or ***prognosis*** of ***breast*** ***cancer*** . The invention further concerns a prognostic method comprising: subjecting a sample of ***breast*** ***cancer*** cells to quant. anal. of the expression of RNA transcript of atleast one gene selected from the group consisting of GRB7, CD68, CTSL, Chk1, AIB1, CCNB1, MCM2, FBXO5, Her2, STK15, EGFR, ***MYBL2*** , HIF1.alpha. and TS, or their product.

L10 ANSWER 19 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:371064 CAPLUS <<LOGINID::20071005>>
DN 140:373461
TI Evaluation of breast cancer states and outcomes using gene expression profiles
IN West, Mike; Nevins, Joseph R.; Huang, Andrew
PA Synpac, Inc., USA; Duke University
SO PCT Int. Appl., 799 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 5

PATENT NO. KIND DATE APPLICATION NO. DATE

PI WO 2004037996 A2 20040506 WO 2003-US33656 20031024
WO 2004037996 A3 20041229
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
US 2004083084 A1 20040429 US 2002-291878 20021112
WO 2004044839 A2 20040527 WO 2002-US38216 20021112
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
US 2004106113 A1 20040603 US 2002-291886 20021112
AU 2003284880 A1 20040513 AU 2003-284880 20031024
PRAI US 2002-420729P P 20021024
US 2002-421062P P 20021025
US 2002-421102P P 20021025
US 2002-424701P P 20021108
US 2002-424715P P 20021108
US 2002-424718P P 20021108
US 2002-291878 A 20021112
US 2002-291886 A 20021112
US 2002-425256P P 20021112
WO 2002-US38216 A 20021112
WO 2002-US38222 A 20021112
US 2003-448461P P 20030221
US 2003-448462P P 20030221

US 2003-457877P P 20030327
US 2003-458373P P 20030331
WO 2003-US33656 W 20031024
AB The present invention relates generally to a method for evaluating and/or predicting breast cancer states and outcomes by measuring gene and metagene expression levels and integrating such data with clin. risk factors. Genes and metagenes whose expressions are correlated with a particular breast cancer risk factor or phenotype are provided using binary prediction tree modeling. The invention provides 175 genes assocd. with metagene predictors of lymph node metastasis, 216 genes assocd. with metagene predictors of breast cancer recurrence, and 496 metagenes related to breast cancer study. Methods of using the subject genes and metagenes in diagnosis and treatment methods, as well as drug screening methods, etc are also provided. In addn., reagents, media and kits that find use in practicing the subject methods are also provided.

L10 ANSWER 20 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2006:28390 BIOSIS <<LOGINID::20071005>>
DN PREV200600031106
TI Expression of the 21 genes in the Recurrence Score assay and prediction of clinical benefit from tamoxifen in NSABP study B-14 and chemotherapy in NSABP study B-20.
AU Paik, S. [Reprint Author]; Shak, S.; Tang, G.; Kim, C.; Joo, H.; Baker, J.; Cronin, M.; Watson, D.; Braynt, J.; Costantino, J.; Wolmark, N.
CS NSABP, Pittsburgh, PA USA
SO Breast Cancer Research and Treatment, (2004) Vol. 88, No. Suppl. 1, pp. S15.
Meeting Info.: 27th Annual Charles A Coltman San Antonio Breast Cancer Symposium. San Antonio, TX, USA. December 08 -11, 2004.
CODEN: BCTRD6. ISSN: 0167-6806.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 28 Dec 2005
Last Updated on STN: 28 Dec 2005

L10 ANSWER 21 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:754553 CAPLUS <<LOGINID::20071005>>
DN 137:227626
TI Methods for diagnosing and monitoring malignancies by screening gene copy numbers
IN Kuo, Wen-Lin; Polikoff, Daniel; Pinkel, Daniel; Albertson, Donna; Berchuk, Andy; Gray, Joe W.
PA The Regents of the University of California, USA
SO PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002077197	A2	20021003	WO 2002-US9419	20020327
WO 2002077197	A3	20031023		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003077582	A1	20030424	US 2001-819148	20010327
AU 2002303165	A1	20021008	AU 2002-303165	20020327
PRAI US 2001-819148	A	20010327		
WO 2002-US9419	W	20020327		

AB The invention concerns the discovery that an amplification of some genes or an increase in that gene activity and a deletion of some genes or a decrease in that gene activity is a marker for the presence of, progression of, or predisposition to, a ***cancer*** (e.g., ***breast*** ***cancer***). Using this information, this invention provides methods of detecting a predisposition to cancer in an animal. The methods involve (i) providing a biol. sample from an animal (e.g. a human patient); (ii) detecting the level of the genes of the present invention within the biol. sample; and (iii) comparing the level of one or more of said genes with a level of one or more of said genes in a control sample taken from a normal, cancer-free tissue.

L10 ANSWER 22 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:3213 CAPLUS <<LOGINID::20071005>>
DN 135:74810
TI Gene expression profiling of primary ***breast*** ***carcinomas*** using arrays of candidate genes
AU Bertucci, Francois; Houlgatte, Remi; Benziane, Athmane; Granjeaud, Samuel; Adelaide, Jose; Tagett, Rebecca; Loriod, Beatrice; Jacquemier, Jocelyne; Viens, Patrice; Jordan, Bertrand; Birnbaum, Daniel; Nguyen, Catherine
CS Laboratoire de Biologie des Tumeurs, TAGC, Institut Paoli-Calmettes (IPC), Marseille, Fr.
SO Human Molecular Genetics (2000), 9(20), 2981-2991
CODEN: HMGEE5; ISSN: 0964-6906
PB Oxford University Press
DT Journal
LA English
AB ***Breast*** ***cancer*** is characterized by an important histoclin. heterogeneity that currently hampers the selection of the most appropriate treatment for each case. This problem could be solved by the identification of new parameters that better predict the natural history of the disease and its sensitivity to treatment. A large-scale mol. characterization of ***breast*** ***cancer*** could help in this context. Using cDNA arrays, we studied the quant. mRNA expression levels of 176 candidate genes in 34 primary ***breast*** ***carcinomas*** along three directions: comparison of tumor samples, correlations of mol. data with conventional histoclin. prognostic features and gene correlations. The study evidenced extensive heterogeneity of breast tumors at the transcriptional level. A hierarchical clustering algorithm identified two molecularly distinct subgroups of tumors characterized by a different clin. outcome after chemotherapy. This outcome could not have been predicted by the commonly used histoclin. parameters. No correlation was found with the age of patients, tumor size, histol. type and grade. However, expression of genes was differential in tumors with lymph node metastasis and according to the estrogen receptor status; ERBB2 expression was strongly correlated with the lymph node status ($P < 0.0001$) and that of GATA3 with the presence of estrogen receptors ($P < 0.001$). Thus, our results identified new ways to group tumors according to outcome and new potential targets of carcinogenesis. They show that the systematic use of cDNA array testing holds great promise to improve the classification of ***breast*** ***cancer*** in terms of ***prognosis*** and chemosensitivity and to provide new potential therapeutic targets.
RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 23 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 3
AN 2000:401121 CAPLUS <<LOGINID::20071005>>
DN 133:294517
TI Frequent amplification of chromosomal region 20q12-q13 in ovarian cancer
AU Tanner, Minna M.; Grenman, Seija; Koul, Anjila; Johannsson, Oskar; Meltzer, Paul; Pejovic, Tanja; Borg, Ake; Isola, Jorma J.
CS Laboratory of Cancer Genetics, Tampere University and University Hospital, Tampere, FIN-33101, Finland
SO Clinical Cancer Research (2000), 6(5), 1833-1839
CODEN: CCREF4; ISSN: 1078-0432
PB American Association for Cancer Research
DT Journal
LA English
AB DNA amplification at chromosomal region 20q12-q13, which is common in ***breast*** ***cancer*** , has recently been described also in ovarian tumors. We studied the amplification of the recently identified candidate oncogenes in this region in 24 sporadic, 3 familial and 4 hereditary ovarian carcinomas, and in 8 ovarian cancer cell lines. High-level amplification of at least one of the five nonsyntenic regions at 20q12-q13.2 was found in 13 sporadic (54%) and in all four hereditary tumors. Typically, two or more distinct amplicons (sepd. by nonamplified DNA) were found coamplified in various combinations. The regions defined by the AIB1 and PTPN1 genes (at 20q12 and 20q13.1, resp.) were amplified in 25% and 29% of the sporadic tumors, also without simultaneous coamplification of other regions. Amplification of AIB1 (a steroid receptor coactivator gene) was assocd. with estrogen receptor positivity in sporadic ovarian carcinomas ($P = 0.01$) and showed a tendency to correlate with poor ***survival*** of patients. Of the genes amplified in ***breast*** ***cancer*** , the BTAK gene was amplified in 21%, the ***MYBL2*** gene in 17%, and the ZNF217 gene in 12.5% of the sporadic tumors. The high frequency of gene amplification at 20q12-q13.2 suggests that the genes amplified therein may play a central role in the pathogenesis of sporadic and hereditary ovarian carcinoma.
RE.CNT 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 16:41:09 ON 05 OCT 2007)

FILE 'STNGUIDE' ENTERED AT 16:41:16 ON 05 OCT 2007

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 16:41:21 ON 05 OCT 2007
L1 173 S MYBL2 OR MYELOBLASTOSIS ONCOGENE-LIKE 2
L2 37 S L1 AND (BREAST CANCER OR BREAST CARCINO?)
L3 31 DUP REM L2 (6 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 16:53:01 ON 05 OCT 2007

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 16:55:48 ON 05 OCT 2007
L4 352725 S BREAST (3A) (CANCER OR CARCIN?)
L5 62163 S L4 AND (PROGNOSIS OR SURVIV?)
L6 23 S L5 AND GENE PROFILE
L7 3 S L6 AND REVIEW
L8 3 DUP REM L7 (0 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 16:59:31 ON 05 OCT 2007

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 17:01:28 ON 05 OCT 2007
L9 26 S L5 AND MYBL2
L10 23 DUP REM L9 (3 DUPLICATES REMOVED)

=> s cancer and gene profi?
L11 686 CANCER AND GENE PROFI?

=> s l11 and review
L12 102 L11 AND REVIEW

=> s l12 and limit?
L13 15 L12 AND LIMIT?

=> dup rem l13
PROCESSING COMPLETED FOR L13
L14 10 DUP REM L13 (5 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 10 ANSWERS - CONTINUE? Y/(N):y

L14 ANSWER 1 OF 10 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 1
AN 2007:171911 BIOSIS <<LOGINID::20071005>>
DN PREV200700160869
TI WNT signaling in the normal intestine and colorectal ***cancer*** .
AU de Lau, Wim; Barker, Nick; Clevers, Hans [Reprint Author]
CS Netherlands Inst Dev Biol, Biomed Ctr, Hubrecht Lab, Uppsalalaan 8, NL-3584 CT Utrecht, Netherlands
clevers@niob.knaw.nl
SO Frontiers in Bioscience, (JAN 1 2007) Vol. 12, pp. 471-491.
ISSN: 1093-9946.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 7 Mar 2007
Last Updated on STN: 7 Mar 2007
AB The intestinal epithelium is a self-renewing tissue that represents a unique model for studying interconnected cellular processes such as proliferation, differentiation, cell migration and carcinogenesis. This ***review*** covers work from the past decade and highlights the importance of the canonical Wnt pathway in regulating multiple aspects of intestinal homeostasis. Numerous in vivo studies combined with ***gene*** ***profiling*** experiments have shown that Wnt signaling promotes maintenance of epithelial stem cells and early progenitors by driving transcription of genes associated with proliferation. These studies also revealed strong similarities between the genetic program initiated by Wnt signals in normal crypt progenitors and in colorectal ***cancer*** cells. More recently it has become apparent that Wnts do not act alone but rather cooperate with Notch signals in maintaining progenitor cell populations. Processes associated with differentiated epithelial cells also appear to be regulated by Wnt signals. For instance, Paneth cells employ active Wnt signals for terminal differentiation. Moreover, through transcriptional regulation of members of the Eph and Ephrin families, Wnt signaling promotes compartmentalization of epithelial cells along the crypt-villus axis. The Eph/Ephrin system also operates to ***limit*** progression of colorectal ***cancer*** beyond the early stages.

L14 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2
AN 2006:970761 CAPLUS <<LOGINID::20071005>>
DN 145:503635
TI Genomics of hepatitis B and C infections: diagnostic and therapeutic applications of microarray profiling
AU Berzsenyi, Mark D.; Roberts, Stuart K.; Beard, Michael R.
CS Department of Gastroenterology, Alfred Hospital, Victoria, Australia
SO Antiviral Therapy (2006), 11(5), 541-552
CODEN: ANTHFA; ISSN: 1359-6535
PB International Medical Press
DT Journal; General Review
LA English
AB A ***review*** . Microarray profiling offers many potential advances in diagnostic and therapeutic intervention in human disease because of its unparalleled ability to conduct high-throughput anal. of gene expression. However, ***limitations*** of this technique relate in part to issues regarding the various methodologies and exptl. designs as well as difficulties in the interpretation of results. Despite this, microarray profiling has led to a better understanding of the mol. pathogenesis of hepatitis B virus (HBV) and hepatitis C virus (HCV) infection. Key events in clearance and the development of chronicity of HCV have been identified that may prove to have a role in the development of future treatments. In addn., pharmacogenomic studies of interferon-based treatment for chronic HCV and HBV have provided mechanistic insights into the therapeutic action of interferons. These advances have implications with respect to the development of improved therapeutic agents. New biomarkers for ***cancer*** screening and ***gene*** ***profiles*** with prognostic value for survival have also been developed for hepatocellular carcinoma, which frequently complicates chronic viral hepatitis. Thus, microarray profiling offers enormous potential for improvements in antiviral therapy and our understanding of blood-borne viral hepatitis.
RE.CNT 81 THERE ARE 81 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 3 OF 10 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2006333408 EMBASE <<LOGINID::20071005>>
TI Follicular lymphoma international prognostic index.
AU Solal-Celigny P.
CS Dr. P. Solal-Celigny, Centre Jean Bernard, 9 rue Beauverger, 72000 Le Mans, France. p.solal-celigny@centre-jean-bernard.org
SO Current Treatment Options in Oncology, (Jul 2006) Vol. 7, No. 4, pp. 270-275.
Refs: 16
ISSN: 1527-2729 CODEN: CTOOBW
CY United Kingdom
DT Journal; General Review; (Review)
FS 016 Cancer
022 Human Genetics
025 Hematology
037 Drug Literature Index
LA English
SL English
ED Entered STN: 1 Aug 2006
Last Updated on STN: 1 Aug 2006
AB Although numerous treatment approaches are proposed for patients with follicular lymphoma, criteria to help in choosing a treatment for a given patient and for comparing trial results are lacking. Several retrospective studies have analyzed prognostic factors, but their conclusions rely on ***limited*** numbers of patients treated during long periods, and their results are discordant. The Follicular Lymphoma International Prognostic Index was designed from the data recorded over 8 years of nearly 5000 patients registered worldwide. Five factors are used (age, Ann Arbor stage, number of nodal sites, serum lactate dehydrogenase level, and hemoglobin level) to build a three-category index. This index, together with new biologic markers such as ***gene*** ***profiling*** and proteomics, could help provide an optimal treatment option for patients with follicular lymphoma. Copyright .COPYRGT. 2006 by Current Science Inc.

L14 ANSWER 4 OF 10 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 3
AN 2005:185959 BIOSIS <<LOGINID::20071005>>
DN PREV200500186516
TI Expression profiling by microarrays in colorectal ***cancer*** (***Review***).
AU Shih, Warren; Chetty, Runjan; Tsao, Ming-Sound [Reprint Author]
CS Ontario Canc InstUniv Hlth NetworkDept Pathol, Princess Margaret Hosp, 610 Univ Ave, Toronto, ON, M5G 2M9, Canada
ming.tsao@uhn.on.ca
SO Oncology Reports, (March 2005) Vol. 13, No. 3, pp. 517-524. print.
ISSN: 1021-335X.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 18 May 2005
Last Updated on STN: 18 May 2005
AB Genome-wide ***gene*** ***profiling*** studies using microarrays have the potential to improve diagnosis and treatment of human cancers. Microarrays have identified many genes that are deregulated in colorectal ***cancer*** compared to normal tissue. Groups of genes that are predictive of tumor stage or presence of metastases, hence putatively associated with ***cancer*** progression have also been revealed. Microarray studies have identified genes whose expression are impacted by chemotherapies for colorectal ***cancer*** , thus could potentially be used to predict response to treatments. Unique gene expression profiles have also been used to classify metastases of uncertain origin. The wide application of microarrays generates exciting prospects in translational research. However, to date overlaps of candidate gene lists associated with specific clinical/biological phenotypes remain disturbingly poor between studies. Overfitting, bias, reporting of only the best results, and fidelity of probe annotations could present ***limitations*** for the interpretation of results shown in microarray publications. Making raw data from these microarray experiments publicly available for analysis by other investigators using different analytical algorithms or for in silico studies may facilitate the most thorough mining of data front these expensive studies. Validations of the results using other more precise techniques and at the biological level represent critical follow-up goals for microarray studies.

L14 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:890002 CAPLUS <<LOGINID::20071005>>
DN 143:226830
TI The role of DNA-microarray in translational ***cancer*** research
AU Korfee, Soenke; Eberhardt, Wilfried; Fujiwara, Yasuhiro; Nishio, Kazuto
CS Shien Lab, National Cancer Center Hospital, Tokyo, 104-0045, Japan
SO Current Pharmacogenomics (2005), 3(3), 201-216
CODEN: CPUHAC; ISSN: 1570-1603
PB Bentham Science Publishers Ltd.
DT Journal; General Review
LA English
AB A ***review*** . The overall prognosis for the majority of ***cancer*** patients remains poor. Current conventional strategies in clin. ***cancer*** research are unable to adequately answer a large no. of important unsolved questions. Although some patients achieve

substantial benefits from classical cytotoxic chemotherapy, others will not. The mechanisms behind this phenomenon are still not identified in detail. Furthermore, the activity of promising novel mol. targeting anticancer agents like tyrosine kinase inhibitors is currently not predictable within the individual patient. The biol. background for this clin. and prognostic heterogeneity in behavior is more or less the large individual variation in the biol. nature of tumors within the same classified histol. subgroup. The overall usefulness of conventional histopathol. classifications to adequately predict patient prognosis or response to chemotherapy is ***limited*** . The most promising way to solve this issue is to found clin. research strategies on basic biol. evidence. New genomic technologies have been developed within recent years. These techniques are able to analyze thousands of genes and their expression profiles simultaneously. An increasing no. of investigations has reported applications of these novel technologies within clin. trials settings. The aim of this approach is to identify new subsets of ***cancer*** patients, to improve prediction of their clin. outcome or response to treatment and select new targets for innovative therapeutic drugs based on the findings from gene expression profiles. Results of these gene expression profile studies could potentially lead to more individually tailored systemic ***cancer*** therapy. In the recent years, a remarkable no. of studies based on these techniques have already been reported. Although the published results are clearly impressive and highly promising, a lot of work remains to be done. Moreover, there is a strong need for an increase in reliability and reproducibility of such gene expression profiling techniques and thus introduction of reproducible quality control in the performance of these assays. Although a large no. of issues remain to be clarified prior to a more general application of genomic profiling techniques in clin. ***cancer*** research, this strategy will eventually turn out as a promising approach to improve successful management of ***cancer*** patients.

RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 4
AN 2005:1110491 CAPLUS <<LOGINID::20071005>>
DN 143:323356
TI Prediction of disease progression and therapy response through molecular markers in breast ***cancer***
AU Hauenstein, Evelyn; Kuschel, Bettina; Meindl, Alfons; Schmitt, Manfred; Kiechle, Marion; Harbeck, Nadia
CS Frauenklinik der Technischen Universitat Munchen, Germany
SO Medizinische Genetik (2005), 17(2), 176-182
CODEN: MGENEZ; ISSN: 0936-5931
PB Verlag Medizinischegenetik
DT Journal; General Review
LA German
AB A ***review*** . Breast ***cancer*** is - like other carcinomas - a disease characterized by certain genetic events in the course of carcinogenesis. It is important to gain a better understanding of the nature and time course of those events. Simultaneously, many efforts were made to avoid either over- or under-therapy and to classify tumors better according to their aggressivity and therapy. For achieving a better classification of tumors, the existing established clin. and histopathol. parameters are not sufficient. Regarding the heterogeneity of tumors, multigen analyses offer a promising prospect to establish such ***gene*** ***profiles*** as new prognostic and predictive markers and evaluate them for clin. use. For some RNA-based concepts, clin. studies for validation and therapy are already being planned. Also, first clin. data for DNA-based techniques describing epigenetic phenomena were validated. Their correlation with disease progress and therapy response is significant and reproducible. Still before such mol. tests can be used for everyday clin. practice, certain quality criteria have to be fulfilled. Also as long as there is no reliable methodical and clin. validation of the existing data, the use of multigen analyses in breast carcinomas should be ***limited*** to clin. studies.

RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 7 OF 10 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 5
AN 2006579944 EMBASE <<LOGINID::20071005>>
TI Prognosis of follicular lymphomas.
AU Solal-Celigny P.
CS P. Solal-Celigny, Department of Hematology and Medical Oncology, Centre Jean Bernard, 9 Rue Beauverger, 72000 Le Mans, France. p.solal-celigny@noos.fr
SO Clinical Lymphoma and Myeloma, (Jun 2005) Vol. 6, No. 1, pp. 21-25.
Refs: 32
ISSN: 1557-9190
CY United States
DT Journal; General Review; (Review)
FS 016 Cancer
025 Hematology
029 Clinical and Experimental Biochemistry
LA English
SL English
ED Entered STN: 19 Dec 2006
Last Updated on STN: 19 Dec 2006
AB Although numerous treatment approaches are proposed for patients with follicular lymphoma, criteria to help in choosing a treatment for a given patient and for comparing trial results are lacking. Several retrospective studies have analyzed prognostic factors, but their conclusions rely on ***limited*** numbers of patients treated during long periods, and their results are discordant. The Follicular Lymphoma International Prognostic Index was designed from the data recorded over 8 years of nearly 5000 patients registered worldwide. Five factors are used (age, Ann Arbor stage, number of nodal sites, serum lactate dehydrogenase level, and hemoglobin level) to build a 3-category index. This index, together with new biologic markers such as ***gene*** ***profiling*** and proteomics, could help provide an optimal treatment option for patients with follicular lymphoma.

L14 ANSWER 8 OF 10 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2005012452 EMBASE <<LOGINID::20071005>>
TI DNA array-based ***gene*** ***profiling*** : From surgical specimen to the molecular portrait of ***cancer*** .
AU Mocellin S.; Provenzano M.; Rossi C.R.; Pilati P.; Nitti D.; Lise M.
CS Dr. S. Mocellin, Clinica Chirurgica Generale II, Dipto. di Sci. Oncol. e Chirurgiche, Universita di Padova, Via Giustiniani, 2, 35128 Padova, Italy. mocellins@hotmail.com
SO Annals of Surgery, (Jan 2005) Vol. 241, No. 1, pp. 16-26.
Refs: 116
ISSN: 0003-4932 CODEN: ANSUA5
CY United States
DT Journal; General Review; (Review)
FS 016 Cancer
022 Human Genetics
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
009 Surgery
LA English
SL English
ED Entered STN: 20 Jan 2005
Last Updated on STN: 20 Jan 2005
AB ***Cancer*** is a heterogeneous disease in most respects, including its cellularity, different genetic alterations, and diverse clinical behaviors. Traditional molecular analyses are reductionist, assessing only 1 or a few genes at a time, thus working with a biologic model too specific and ***limited*** to confront a process whose clinical outcome is likely to be governed by the combined influence of many genes. The potential of functional genomics is enormous, because for each experiment, thousands of relevant observations can be made simultaneously. Accordingly, DNA array, like other high-throughput technologies, might catalyze and ultimately accelerate the development of knowledge in tumor cell biology. Although in its infancy, the implementation of DNA array technology in ***cancer*** research has already provided investigators with novel data and intriguing new hypotheses on the molecular cascade leading to carcinogenesis, tumor aggressiveness, and sensitivity to antiblastic agents. Given the revolutionary implications that the use of this technology might have in the clinical management of patients with ***cancer*** , principles of DNA array-based tumor ***gene*** ***profiling*** need to be clearly understood for the data to be correctly interpreted and appreciated. In the present work, we discuss the technical features characterizing this powerful laboratory tool and ***review*** the applications so far described in the field of oncology.

L14 ANSWER 9 OF 10 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2004207361 EMBASE <<LOGINID::20071005>>
TI Integration of multimodality approaches in the management of malignant pleural mesothelioma.
AU Martino D.; Pass H.I.
CS Dr. H.I. Pass, Harper University Hospital, 3990 John R, Detroit, MI 48201, United States. hpass@dmc.org
SO Clinical Lung Cancer, (Mar 2004) Vol. 5, No. 5, pp. 290-298.
Refs: 95
ISSN: 1525-7304 CODEN: CLCLCA
CY United States
DT Journal; General Review; (Review)
FS 014 Radiology
015 Chest Diseases, Thoracic Surgery and Tuberculosis
016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
039 Pharmacy
LA English
SL English
ED Entered STN: 4 Jun 2004
Last Updated on STN: 4 Jun 2004
AB More than half a century after the first descriptions of mesothelioma as a pathologic entity, satisfactory treatment is still elusive. Although relatively uncommon, the incidence of mesothelioma will most likely increase over the next 10-20 years. Advances have been made in understanding the pathogenesis, diagnosis, and staging, but they have not translated into markedly improved survival. Some use palliative treatment as the primary means of therapy even now. On the other hand, a cadre of individuals have studied how surgery, chemotherapy, and radiation therapy affect the disease. Although each individual modality has had ***limited*** success by itself, a multimodality approach has been documented to improve survival and quality of life. In addition, intriguing discoveries in immunology and ***gene*** ***profiling*** and therapy promise hope for further improvement. In this article, we

will illustrate the current views on integrating these different approaches and delineate areas of active research.

L14 ANSWER 10 OF 10 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003110299 EMBASE <<LOGINID::20071005>>
TI The unknown biology of the unknown primary tumour: A literature ***review*** .
AU van de Wouw A.J.; Jansen R.L.H.; Speel E.J.M.; Hillen H.F.P.
CS Dr. A.J. van de Wouw, Department of Internal Medicine, Slingeland Hospital Doetinchem, PO Box 169, 7000 AD Doetinchem, Netherlands. y.van.de.wouw@slingeland.nl
SO Annals of Oncology, (1 Feb 2003) Vol. 14, No. 2, pp. 191-196.
Refs: 75
ISSN: 0923-7534 CODEN: ANONE2
CY United Kingdom
DT Journal; General Review; (Review)
FS 016 Cancer
022 Human Genetics
029 Clinical and Experimental Biochemistry
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
LA English
SL English
ED Entered STN: 27 Mar 2003
Last Updated on STN: 27 Mar 2003
AB The unknown primary tumour (UPT) is an intriguing clinical phenomenon found in approximately 5% of all newly diagnosed patients with ***cancer*** . It is unclear whether UPT forms a distinct biological entity with specific genetic and phenotypic characteristics, or whether it is merely a clinical presentation of metastases in patients in whom the primary tumour cannot be detected and does not result in any visible clinical signs. Understanding the basic biology of UPT may shed light on this issue and, moreover, may have a direct impact on clinical care. A ***review*** of the literature revealed only a ***limited*** number of publications describing the genetic and phenotypic features of UPT, most of which focus only on the potential of these markers to predict prognosis. The question as to whether the biology of UPT is different from tumours of known primaries therefore remains unanswered. Further insight into the molecular mechanisms underlying the oncogenesis of UPT, e.g. by applying newly available DNA and ***gene*** ***profiling*** microarray techniques, will be necessary to understand its specific biology and to develop more effective treatments.

=> d his

(FILE 'HOME' ENTERED AT 16:41:09 ON 05 OCT 2007)

FILE 'STNGUIDE' ENTERED AT 16:41:16 ON 05 OCT 2007

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 16:41:21 ON 05 OCT 2007
L1 173 S MYBL2 OR MYELOBLASTOSIS ONCOGENE-LIKE 2
L2 37 S L1 AND (BREAST CANCER OR BREAST CARCINO?)
L3 31 DUP REM L2 (6 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 16:53:01 ON 05 OCT 2007

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 16:55:48 ON 05 OCT 2007
L4 352725 S BREAST (3A) (CANCER OR CARCIN?)
L5 62163 S L4 AND (PROGNOSIS OR SURVIV?)
L6 23 S L5 AND GENE PROFILE
L7 3 S L6 AND REVIEW
L8 3 DUP REM L7 (0 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 16:59:31 ON 05 OCT 2007

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 17:01:28 ON 05 OCT 2007
L9 26 S L5 AND MYBL2
L10 23 DUP REM L9 (3 DUPLICATES REMOVED)
L11 686 S CANCER AND GENE PROFI?
L12 102 S L11 AND REVIEW
L13 15 S L12 AND LIMIT?
L14 10 DUP REM L13 (5 DUPLICATES REMOVED)

=> s l12 and (progno? or surviv?)
L15 54 L12 AND (PROGNO? OR SURVIV?)

=> dup rem l15
PROCESSING COMPLETED FOR L15
L16 43 DUP REM L15 (11 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 43 ANSWERS - CONTINUE? Y/(N):y

L16 ANSWER 1 OF 43 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2007:374344 BIOSIS <<LOGINID::20071005>>
DN PREV200700374919
TI Gene expression profiles as biomarkers for the prediction of chemotherapy drug response in human tumour cells.
AU Parissenti, Amadeo M. [Reprint Author]; Hembruff, Stacey L.; Villeneuve, David J.; Veitch, Zachary; Guo, Baoqing; Eng, Jamei
CS Sudbury Reg Hosp, Tumor Biol Res Program, 41 Ramsey Lake Rd, Sudbury, ON P3E 5J1, Canada
aparissenti@hrsrh.on.ca
SO Anti-Cancer Drugs, (JUN 2007) Vol. 18, No. 5, pp. 499-523.
CODEN: ANTDEV. ISSN: 0959-4973.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 4 Jul 2007
Last Updated on STN: 4 Jul 2007
AB Genome profiling approaches such as cDNA microarray analysis and quantitative reverse transcription polymerase chain reaction are playing ever-increasing roles in the classification of human cancers and in the discovery of biomarkers for the prediction of ***prognosis*** in ***cancer*** patients. Increasing research efforts are also being directed at identifying set of genes whose expression can be correlated with response to specific drugs or drug combinations. Such genes hold the prospect of tailoring chemotherapy regimens to the individual patient, based on tumour or host gene expression profiles. This ***review*** outlines recent advances and challenges in using genome profiling for the identification of tumour or host genes whose expression correlates with response to chemotherapy drugs both in vitro and in clinical studies. Genetic predictors of response to a variety of anticancer agents are discussed, including the anthracyclines, taxanes, topoisomerase I and II inhibitors, nucleoside anatogs, alkylating agents, and vinca alkaloids.

L16 ANSWER 2 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2007464172 EMBASE <<LOGINID::20071005>>
TI Development of farnesyltransferase inhibitors for clinical ***cancer*** therapy: Focus on hematologic malignancies.
AU Karp J.E.; Lancet J.E.
CS Dr. Prof. J.E. Karp, Leukemia Program, Division of Hematologic Malignancies, Johns Hopkins Sidney Kimmel Comprehensive Cancer Center, 1650 Orleans Street, Baltimore, MD 21231-1000, United States. jkarp2@jhmi.edu
SO Cancer Investigation, (Sep 2007) Vol. 25, No. 6, pp. 484-494.
Refs: 95
ISSN: 0735-7907 E-ISSN: 1532-4192 CODEN: CINVD7
PUI 782021553
CY United States
DT Journal; General Review; (Review)
FS 016 Cancer
025 Hematology
037 Drug Literature Index
038 Adverse Reactions Titles
006 Internal Medicine
LA English
SL English
ED Entered STN: 2 Oct 2007
Last Updated on STN: 2 Oct 2007
AB Farnesyltransferase inhibitors (FTIs) target and inhibit the peptide prenylating enzyme farnesyltransferase. This new class of signal transduction inhibitors is being tested clinically in diverse malignancies, with encouraging results in hematololgic malignancies and breast ***cancer*** in particuarl. Critical questions have yet to be answered, for example, optimal dose and schedule, disease subgroups most likely to respond, and appropriate combinations with standard cytotoxics and new biologics. ***Gene*** ***profiling*** studies of malignant target cells obtained during FTI clinical trials will help to identify patients who are likely to respond to FTIs and to develop mechanisms for overcoming FTI resistance. Clinical trials and correlative laboratory studies in progress and under development will define the optimal roles of FTIs in ***cancer*** patients. Copyright .COPYRGT. Informa Healthcare USA, Inc.

L16 ANSWER 3 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2007267613 EMBASE <<LOGINID::20071005>>
TI Biomarkers for mesothelioma.
AU Scherpereel A.; Lee Y.C.G.
CS Dr. Y.C.G. Lee, Centre for Respiratory Research, University College London, 5 University Street, London WC1E 6JJ, United Kingdom. ycgarylee@hotmail.com
SO Current Opinion in Pulmonary Medicine, (Jul 2007) Vol. 13, No. 4, pp. 339-343.
Refs: 48
ISSN: 1070-5287 E-ISSN: 1531-6971 CODEN: COPMFY
PUI 000631982007070000018
CY United States
DT Journal; General Review; (Review)
FS 016 Cancer
017 Public Health, Social Medicine and Epidemiology
022 Human Genetics
029 Clinical and Experimental Biochemistry
005 General Pathology and Pathological Anatomy
LA English
SL English
ED Entered STN: 14 Jun 2007
Last Updated on STN: 14 Jun 2007
AB PURPOSE OF ***REVIEW*** : Mesothelioma is an incurable ***cancer*** and its global incidence continues to increase. There has been strong

interest in the search for a biomarker that would be of value for the diagnosis, ***prognosis*** and disease monitoring of mesothelioma. Large series evaluating the use of novel candidate markers have recently been published. RECENT FINDINGS: To date, global ***gene*** ***profiling*** studies have failed to find a molecule that reliably captures all subtypes of mesothelioma, and differentiates it from benign pathologies and metastatic carcinomas. Soluble mesothelin-related peptide (SMRP), osteopontin and megakaryocyte potentiating factor have been assessed as markers. SMRP testing is clinically available and provides reasonable diagnostic sensitivity and specificity when applied to serum or pleural fluid. Elevated SMRP levels can occur in metastatic, especially ovarian and pancreatic, adenocarcinomas. False negatives are common with sarcomatoid mesothelioma. SMRP levels may reflect tumor load and disease progression. The role of SMRP in predicting mesothelioma development in subjects exposed to asbestos has raised interest. Osteopontin lacks specificity as a diagnostic marker for mesothelioma but may have value in disease monitoring. SUMMARY: The proposed markers have insufficient accuracy to replace cytohistology as the gold standard for diagnosis for mesothelioma. Elevated SMRP levels raise suspicion of mesothelioma although negative values do not exclude disease. Its role in disease monitoring in patients and in predicting disease development in at-risk individuals warrant further study. .COPYRGT. 2007 Lippincott Williams & Wilkins, Inc.

L16 ANSWER 4 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2007312628 EMBASE <<LOGINID::20071005>>
TI Biological characterization of the basal-like subtype of invasive breast carcinoma.
AU Yu H.; Hu X.-C.
CS H. Yu, Department of Medical Oncology, Cancer Hospital, Fudan University, Shanghai 200031, China
SO Chinese Journal of Cancer Prevention and Treatment, (Feb 2007) Vol. 14, No. 3, pp. 234-236.
Refs: 22
ISSN: 1673-5269
CY China
DT Journal; General Review; (Review)
FS 016 Cancer
LA Chinese
SL Chinese; English
ED Entered STN: 1 Aug 2007
Last Updated on STN: 1 Aug 2007
AB Recent ***gene*** ***profiling*** studies classify breast carcinomas into five groups: estrogen receptor(ER)+/luminalA, luminalB (ER+), normal breast-like groups, Her-2 overexpressing, and basal-like groups, with the latter two associated with poor outcomes. Being differ from Her-2 over-expressing, the biological characterization of the basal-like subtype of invasive breast carcinoma is not known clearly. Using gene expression profiles, we find that this subtype is typically immunohistochemically negative for estrogen receptor and Her-2 but positive for basal cytokeratin 5/6, cytokeratin 17 and HER1, and is correlative to BRCA1 mutations. There is a small heat shock protein named as a-basic-crystallin, as an oncogene, overexpression leads to lose apoptosis of mammary epithelial cells, which inhibits the pathway of MAPK/REK kinase. Then we can suppose that the .alpha.-basic-crystallin is a potential target for these tumors.

L16 ANSWER 5 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2007256910 EMBASE <<LOGINID::20071005>>
TI Individualization of therapy using mammaprint.RTM..RTM.: From development to the MINDACT trial.
AU Mook S.; Van't Veer L.J.; Rutgers E.J.T.; Piccart-Gebhart M.J.; Cardoso F.
CS Dr. F. Cardoso, Medical Oncology and Translational Research, Jules Bordet Institute, Boulevard de Waterloo, 125, 1000 Brussels, Belgium. fatima.cardoso@bordet.be
SO Cancer Genomics and Proteomics, (May 2007) Vol. 4, No. 3, pp. 147-156.
Refs: 28
ISSN: 1109-6535
CY Greece
DT Journal; General Review; (Review)
FS 016 Cancer
022 Human Genetics
027 Biophysics, Bioengineering and Medical Instrumentation
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
ED Entered STN: 12 Jun 2007
Last Updated on STN: 12 Jun 2007
AB To date, most treatment decisions for adjuvant chemotherapy in breast ***cancer*** are based on conventional clinicopathological criteria. Since breast ***cancer*** tumors with similar clinicopathological characteristics can have strikingly different outcomes, the current selection for adjuvant chemotherapy is far from accurate. Using high-throughput microarray analysis, a 70-gene signature was identified which can accurately select early stage breast ***cancer*** patients who are highly likely to develop distant metastases, and therefore, may benefit the most from adjuvant chemotherapy. This ***review*** describes the development of the 70- ***gene*** ***profile*** (Mammaprint.RTM..RTM.), its retrospective validation and feasibility studies, and its prospective validation in the large adjuvant MINDACT (Microarray In Node-negative Disease may Avoid ChemoTherapy) clinical trial.

L16 ANSWER 6 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2007:123019 CAPLUS <<LOGINID::20071005>>
DN 147:44833
TI Clinical ***cancer*** advances 2006: Major Research Advances in ***Cancer*** Treatment, Prevention, and Screening - A Report From the American Society of Clinical Oncology
AU Ozols, Robert F.; Herbst, Roy S.; Colson, Yolonda L.; Gralow, Julie; Bonner, James; Curran, Walter J., Jr.; Eisenberg, Burton L.; Ganz, Patricia A.; Kramer, Barnett S.; Kris, Mark G.; Markman, Maurie; Mayer, Robert J.; Raghavan, Derek; Reaman, Gregory H.; Sawaya, Raymond; Schilsky, Richard L.; Schuchter, Lynn M.; Sweetenham, John W.; Vahdat, Linda T.; Winn, Rodger J.
CS American Society of Clinical Oncology, Alexandria, VA, USA
SO Journal of Clinical Oncology (2007), 25(1), 146-162
CODEN: JCONDN; ISSN: 0732-183X
PB American Society of Clinical Oncology
DT Journal; General Review
LA English
AB A ***review*** . A report on the most significant advances on the front lines of ***cancer*** research is presented. Such advances include the approval of the world's first preventive vaccine for human papillomavirus (HPV), a virus strongly assocd. with cervical ***cancer*** ; the effective new targeted therapies for hard-to-treat cancers such as kidney ***cancer*** , HER-2 pos. breast ***cancer*** , chronic myelogenous leukemia resistant to the current std. treatment, and head and neck ***cancer*** ; and the creation of a novel ***gene*** ***profiling*** test to predict lung ***cancer*** ***prognosis*** .
RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 7 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2007:762866 CAPLUS <<LOGINID::20071005>>
DN 147:267963
TI Clinical relevance of the inhibitory effect of Green Tea Catechins (GTCs) on prostate ***cancer*** progression in combination with molecular profiling of catechin-resistant tumors: an integrated view
AU Bettuzzi, S.; Rizzi, F.; Belloni, L.
CS Department of Experimental Medicine, University of Parma, Parma, 43-100, Italy
SO Polish Journal of Veterinary Sciences (2007), 10(1), 57-60
CODEN: PJVSFK; ISSN: 1505-1773
PB Polish Academy of Sciences, Committee of Veterinary Sciences
DT Journal; General Review
LA English
AB A ***review*** . Prostate ***cancer*** (CaP) is a fast-growing health and social problem already representing the second leading cause of ***cancer*** -related death among men in Western countries. Lifestyle-related factors and diet are major contributors for CaP promotion. Because of unfavorable ***prognosis*** of extra-prostatic CaP, prevention is considered the best approach to fight it at present time. Green Tea Catechins (GTCs) were proven effective at inhibiting ***cancer*** growth in several lab. studies. We recently performed a pilot clin. trial in HG-PIN subjects showing that only 1/30 tumor was diagnosed in subjects treated for 1 yr with 600 mg/die GTCs, while 9/30 cancers were found in placebo-treated men. CaP is an elusive disease, whose biol. behavior is difficult to predict. We have recently described and validated a RT-qPCR method based on a 8-genes signature that significantly discriminated benign tissue from CaP in both humans and TRAMP mice spontaneously developing CaP. In the animal model, also GTCs-resistant CaP was significantly discriminated from GTCs-sensitive CaP, i.e. responding to GTCs administration. Preliminary expts. in our lab. have shown that this method can be successfully applied to a single tissue needle biopsy specimen in humans. The combination of these results may be of particular significance on the field. In fact, GTCs treatment for men at high risk of CaP as first line prevention therapy in combination with the 8-genes signature profiling in tissue needle biopsies for real time monitoring of patient's response might importantly change, in the near future, the clin. managing of this highly diffuse malignancy.
RE.CNT 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 8 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1
AN 2006:1227781 CAPLUS <<LOGINID::20071005>>
DN 146:313633
TI Mechanisms of disease: adrenocortical tumors-molecular advances and clinical perspectives
AU Bertherat, Jerome; Groussin, Lionel; Bertagna, Xavier
CS Cochin Hospital, University of Paris 5, Paris, Fr.
SO Nature Clinical Practice Endocrinology & Metabolism (2006), 2(11), 632-641
CODEN: NCPEDB; ISSN: 1745-8366
PB Nature Publishing Group
DT Journal; General Review
LA English
AB A ***review*** . Most adrenocortical tumors are benign, unilateral, adrenocortical adenomas that are often discovered incidentally. Adrenocortical ***cancer*** is rare. Exceptionally, adrenocortical

tumors can be bilateral. Although most adrenocortical tumors occur sporadically, they may also feature in congenital and/or familial disease. The identification of germline genetic defects in familial diseases assocd. with adrenocortical tumors helped to define the somatic alterations in sporadic disease: for example, overexpression of insulin-like growth factor 2 and alterations at the 11p15 locus (obsd. in Beckwith-Wiedemann syndrome) are also found in most adrenocortical cancers. Similarly, inactivating mutations of the TP53 gene, located at 17p13 (obsd. in Li-Fraumeni syndrome), can also be found at the somatic level in sporadic adrenocortical cancers, as can 17p13 allelic losses. Components of the cAMP signaling pathway-for example, adrenocorticotropic hormone receptors and other membrane receptors, Gs proteins and protein kinase A-can be altered to various degrees in adrenocortical tumors. More recently, ***gene*** ***profiling*** and genetic studies have shown that the Wnt-.beta.-catenin signaling pathway is frequently activated in adrenocortical tumors. These research findings already have profound implications for clin. management of patients with adrenocortical tumors, for example in unraveling the genetic origin of the disease in some patients, and in the development of mol. markers for diagnosis and ***prognosis*** . The new findings should also help in the development of new therapeutic options.
RE.CNT 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 9 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2
AN 2006:970761 CAPLUS <<LOGINID::20071005>>
DN 145:503635
TI Genomics of hepatitis B and C infections: diagnostic and therapeutic applications of microarray profiling
AU Berzsenyi, Mark D.; Roberts, Stuart K.; Beard, Michael R.
CS Department of Gastroenterology, Alfred Hospital, Victoria, Australia
SO Antiviral Therapy (2006), 11(5), 541-552
CODEN: ANTHFA; ISSN: 1359-6535
PB International Medical Press
DT Journal; General Review
LA English
AB A ***review*** . Microarray profiling offers many potential advances in diagnostic and therapeutic intervention in human disease because of its unparalleled ability to conduct high-throughput anal. of gene expression. However, limitations of this technique relate in part to issues regarding the various methodologies and exptl. designs as well as difficulties in the interpretation of results. Despite this, microarray profiling has led to a better understanding of the mol. pathogenesis of hepatitis B virus (HBV) and hepatitis C virus (HCV) infection. Key events in clearance and the development of chronicity of HCV have been identified that may prove to have a role in the development of future treatments. In addn., pharmacogenomic studies of interferon-based treatment for chronic HCV and HBV have provided mechanistic insights into the therapeutic action of interferons. These advances have implications with respect to the development of improved therapeutic agents. New biomarkers for ***cancer*** screening and ***gene*** ***profiles*** with ***prognostic*** value for ***survival*** have also been developed for hepatocellular carcinoma, which frequently complicates chronic viral hepatitis. Thus, microarray profiling offers enormous potential for improvements in antiviral therapy and our understanding of blood-borne viral hepatitis.
RE.CNT 81 THERE ARE 81 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 10 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2006579739 EMBASE <<LOGINID::20071005>>
TI Urinary bladder tumor markers.
AU Lokeshwar V.B.; Selzer M.G.
CS Dr. V.B. Lokeshwar, Department of Urology, University of Miami Miller School of Medicine, Miami, FL 33101, United States. vlokeshw@med.miami.edu
SO Urologic Oncology: Seminars and Original Investigations, (Nov 2006) Vol. 24, No. 6, pp. 528-537.
Refs: 85
ISSN: 1078-1439 CODEN: UOSOAA
PUI S 1078-1439(06)00147-5
CY United States
DT Journal; General Review; (Review)
FS 017 Public Health, Social Medicine and Epidemiology
022 Human Genetics
028 Urology and Nephrology
036 Health Policy, Economics and Management
LA English
SL English
ED Entered STN: 14 Dec 2006
Last Updated on STN: 14 Dec 2006
AB Bladder ***cancer*** is amenable to biomarker development because many tumor-associated molecules are secreted in urine. Tumor cells are shed in urine, and, therefore, tests that detect tumor cell-surface markers have also been developed to diagnose bladder ***cancer*** and monitor its recurrence. Several bladder tumor markers show higher sensitivity than cytology, but most have lower specificity. In addition to markers that use conventional technologies such as enzyme-linked immunosorbent assay, point-of-care devices, reverse transcriptase polymerase chain reaction, fluorescent in situ hybridization, and immunocytochemistry, proteomic and ***gene*** ***profiling*** approaches are being used to find new biomarkers to assist in the molecular profiling of bladder ***cancer*** . This ***review*** describes both new and well-studied bladder tumor markers. .COPYRGT. 2006 Elsevier Inc. All rights reserved.

L16 ANSWER 11 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 3
AN 2006:481681 CAPLUS <<LOGINID::20071005>>
DN 146:115387
TI Microarrays in gastrointestinal ***cancer*** : is personalized prediction of response to chemotherapy at hand?
AU Jensen, Eric H.; McLoughlin, James M.; Yeatman, Timothy J.
CS H. Lee Moffitt Cancer Center and Research Institute, Tampa, FL, USA
SO Current Opinion in Oncology (2006), 18(4), 374-380
CODEN: CUOOE8; ISSN: 1040-8746
PB Lippincott Williams & Wilkins
DT Journal; General Review
LA English
AB A ***review*** . Mol. profiling has proven to be an invaluable tool in ***cancer*** research. Although only in its infancy, microarray technol. and gene arrays have led to substantial advances in tumor identification, staging and prediction of response. This ***review*** outlines some of the more recent advances in the use of microarrays as a novel means to advance the std. of care for patients with gastrointestinal cancers. Recent findings: Recent investigations have shown that gene expression profiles can be used to identify, stage, and guide therapeutic intervention in many gastrointestinal cancers. In cases of unknown primary disease, genetic fingerprints can be used to define the origin of the tumor in the majority of cases. Similarly, gene expression has been shown to allow for more accurate staging of patients with a variety of tumor types. Perhaps most exciting is early data that support the potential for microarray to guide therapeutic intervention by providing specific gene fingerprints which correlate with sensitivity to specific chemotherapy, biol. therapy, or other ***cancer*** treatments. Summary: Gene microarrays have become a powerful resource in ***cancer*** investigations. Individualized ***cancer*** care based on specific ***gene*** ***profiles*** is on the horizon for patients with gastrointestinal cancers.
RE.CNT 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 12 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2006333408 EMBASE <<LOGINID::20071005>>
TI Follicular lymphoma international ***prognostic*** index.
AU Solal-Celigny P.
CS Dr. P. Solal-Celigny, Centre Jean Bernard, 9 rue Beauverger, 72000 Le Mans, France. p.solal-celigny@centre-jean-bernard.org
SO Current Treatment Options in Oncology, (Jul 2006) Vol. 7, No. 4, pp. 270-275.
Refs: 16
ISSN: 1527-2729 CODEN: CTOOBW
CY United Kingdom
DT Journal; General Review; (Review)
FS 016 Cancer
022 Human Genetics
025 Hematology
037 Drug Literature Index
LA English
SL English
ED Entered STN: 1 Aug 2006
Last Updated on STN: 1 Aug 2006
AB Although numerous treatment approaches are proposed for patients with follicular lymphoma, criteria to help in choosing a treatment for a given patient and for comparing trial results are lacking. Several retrospective studies have analyzed ***prognostic*** factors, but their conclusions rely on limited numbers of patients treated during long periods, and their results are discordant. The Follicular Lymphoma International ***Prognostic*** Index was designed from the data recorded over 8 years of nearly 5000 patients registered worldwide. Five factors are used (age, Ann Arbor stage, number of nodal sites, serum lactate dehydrogenase level, and hemoglobin level) to build a three-category index. This index, together with new biologic markers such as ***gene*** ***profiling*** and proteomics, could help provide an optimal treatment option for patients with follicular lymphoma. Copyright .COPYRGT. 2006 by Current Science Inc.

L16 ANSWER 13 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2007:386686 CAPLUS <<LOGINID::20071005>>
TI Clinical utility of microarray-derived genetic signatures in predicting outcomes in prostate ***cancer***
AU Reddy, G. Kesava; Balk, Steven P.
CS CIG Media Group, LP, Dallas, TX, USA
SO Clinical Genitourinary Cancer (2006), 5(3), 187-189
CODEN: CGCLA7; ISSN: 1558-7673
PB Cancer Information Group
DT Journal; General Review
LA English
AB Prostate ***cancer*** is a complex heterogeneous disease, and risk stratification remains a significant clin. challenge. Gene microarray has been developed to provide better prediction of clin. outcomes and potentially improve management of patients with various malignancies, including prostate ***cancer*** . Currently, several studies are

evaluating the clin. significance of gene expression signatures in prostate ***cancer*** . These approaches might provide outcome predictions, such as treatment response, progression-free ***survival*** , overall ***survival*** , and metastatic status and offer new strategies to identify patients at high risk for personalized ***cancer*** therapies. This article discusses the latest developments in gene expression-based signatures that predict clin. behavior of prostate ***cancer*** . ***Gene*** ***profiling*** could lead to enhanced early detection and ***prognosis*** of prostate ***cancer*** , resulting in improved overall ***survival*** . The ability to predict clin. outcomes by the microarray-derived genetic signatures is promising; however, further studies are warranted to optimize its clin. utility in patients with prostate ***cancer*** .
RE.CNT 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 14 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2006509934 EMBASE <<LOGINID::20071005>>
TI Clinical relevance of alternative splicing.
AU Ravindra T.; Lakshmi N.K.; Chaitanya K.; Surender V.; Ahuja Y.R.
CS T. Ravindra, UGC Research Unit, Bhavans New Science College, Narayanguda,
Hyderabad - 500 029, India. rravindra_tiwari@yahoo.com
SO Indian Journal of Human Genetics, (1 Jun 2006) Vol. 12, No. 2, pp. 45-52.
Refs: 77
ISSN: 0971-6866 CODEN: IJHGA2
CY India
DT Journal; General Review; (Review)
FS 022 Human Genetics
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LA English
SL English
ED Entered STN: 14 Nov 2006
Last Updated on STN: 14 Nov 2006
AB The unique phenomenon of alternative splicing is gathering concern due to its promising therapeutic potential. The human genome sequencing project suggests approximately 20,000-25,000 genes. Among these, about 35-60% of genes generate multiple mRNAs by alternative splicing mechanism and contribute to the diversity of the proteomic world. This 'gene shortfall' has ignited considerable interest in alternative RNA splicing. This process leads to expression of a single gene responsible for the transcription of different mRNA isoforms that might have multiple biological functions. The disruption of splicing pattern can produce aberrant splice variants, which are implicated in more than 50% of genetic disorders including ***cancer*** . Altered splice sites in neoplastic cell contribute to the development, progression and/or maintenance of tumorous growth. The repertoire of tumor-specific variant represents a potential marker in pharmacogenomic diagnostic relevance. Alternative splice isoforms have been analyzed serendipitously by qualitative ***gene*** ***profiling*** with in silico gene prediction software. Computational approach in identifying exonic splicing enhancers in genomic DNA and focus on microarray technology will elucidate differential expression of alternative splice variants. The antisense oligonucleotides modulate alternative splicing and engender the production of therapeutic gene products. Oligonucleotides have the potential to silence the mutations caused by aberrant splicing. The efficacy of the antisense oligonucleotides lies in the chemical configuration, affinity and delivery strategies. Hence the therapeutic potential of antisense oligonucleotides as modulators of aberrant alternative splicing would be a major challenge to the upcoming proteomic era.

L16 ANSWER 15 OF 43 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN DUPLICATE 4
AN 2005:522359 BIOSIS <<LOGINID::20071005>>
DN PREV200510298141
TI Molecular ***prognostic*** markers in pancreatic ***cancer*** : A systematic ***review*** .
AU Garcea, G. [Reprint Author]; Neal, C. P.; Pattenden, C. J.; Steward, W. P.; Berry, D. P.
CS Univ Leicester, Leicester Royal Infirm, Robert Kilpatrick Clin Sci Bldg, Leicester LE2 7LX, Leics, UK
gg43@le.ac.uk
SO European Journal of Cancer, (OCT 2005) Vol. 41, No. 15, pp. 2213-2236.
CODEN: EJCAEL. ISSN: 0959-8049.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 23 Nov 2005
Last Updated on STN: 23 Nov 2005
AB Pancreatic ***cancer*** is one of the most lethal tumours of the gastrointestinal tract. The ability to predict which patients would benefit most from surgical intervention and/or chemotherapy would be a great clinical asset. Considerable research has focused on identifying molecular events in pancreatic carcinogenesis, and their correlation with clinicopathological variables of pancreatic tumours and ***survival*** . This systematic ***review*** examined evidence from published manuscripts looking at molecular markers in pancreatic ***cancer*** and their correlation with tumour stage and grade, response to chemotherapy and long-term ***survival*** . A literature search was undertaken using PubMed and MEDLINE search engines, using the keywords p53, p21, p16, p27, SMAD4, K-ras, cyclin D1, Bax, Bcl-2, EGFR, EGF, c-erbB2, HB-EGF, TGF beta, FGF, MMP, uPA, cathepsin, heparanase, E-cadherin, laminins, integrins, TMSF, CD44, cytokines, angiogenesis, VEGF, IL-8, beta-catenin, DNA microarray, and ***gene*** ***profiling*** . A bewildering number of biomarkers are currently under evaluation. For the most part, the evidence regarding their application as ***prognostic*** indicators is conflicting. The advent of gene microarray and mass spectrometric protein profiling offers the potential to examine many different biomarkers simultaneously. This 'protein/gene signature' could revolutionise work in this field and allow researchers to develop accurate and reproducible predictions of ***survival*** based on protein or ***gene*** ***profiles*** . (c) 2005 Elsevier Ltd. All rights reserved.

L16 ANSWER 16 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:304984 CAPLUS <<LOGINID::20071005>>
DN 143:323210
TI Gene expression profiling of breast ***cancer*** : a new tumor marker
AU van't Veer, Laura J.; Paik, Soonmyung; Hayes, Daniel F.
CS Division of Diagnostic Oncology, The Netherlands Cancer Institute, Amsterdam, Neth.
SO Journal of Clinical Oncology (2005), 23(8), 1631-1635
CODEN: JCONDN; ISSN: 0732-183X
PB American Society of Clinical Oncology
DT Journal; General Review
LA English
AB A ***review*** . A ***review*** describes the multiplex, real-time reverse transcriptase PCR assays, which have been developed that permit interrogation of several hundred genes using unlimited amts. of formalin-fixed, paraffin-embedded sections. The concept of ***gene*** ***profiling*** and development of signature pattern recognition, which may provide a powerful tool to identify ***prognosis*** and likelihood of benefit or resistance to selected therapeutic agents, is discussed.
RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 17 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 5
AN 2006:276338 CAPLUS <<LOGINID::20071005>>
DN 144:448494
TI ***Prognostic*** factors in follicular lymphomas
AU Solal-Celigny, Philippe; Cartron, Gilles
CS Centre Jean Bernard, Le Mans, 72000, Fr.
SO Hematologie (2005), 11(6), 417-424
CODEN: HEMAF2; ISSN: 1264-7527
PB John Libbey Eurotext
DT Journal; General Review
LA French
AB A ***review*** . With conventional treatments, the course of patients with follicular lymphoma was fatal in most cases. These treatments did not significantly modify the natural course of the disease. For the last ten years, innovative approaches have been proposed using interferon .alpha., anti CD 20 monoclonal antibodies, radiolabeled or not, stem cell transplantation. The morbidity and the cost of these treatments are highly variable and the choice of a treatment must rely on objective parameters. Recently, several ***prognostic*** factors have been investigated in follicular lymphomas. Grade 3 follicular lymphomas with a poor ***prognosis*** have been individualized by pathologists. Using 5 parameters (age, no. of nodal sites involved, Ann Arbor stage, serum LDH level, Hb level), the Follicular Lymphoma International ***Prognostic*** Index allows to sep. patients into three risk groups with significantly different hazard-ratios for death. Cytogenetic studies, and esp. some addnl. abnormalities to the t(14;18) translocation, and more recently, mol. and ***gene*** ***profile*** analyses add to this ***prognostic*** information.
RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 18 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2005576108 EMBASE <<LOGINID::20071005>>
TI Artificial intelligence in hematology.
AU Zini G.
CS G. Zini, Catholic University of Sacred Heart, Hematology, Laboratory, Policlinico Gemelli, L.go Gemelli 8, 00168 Rome, Italy.
recamh@rm.unicatt.it
SO Hematology, (Oct 2005) Vol. 10, No. 5, pp. 393-400.
Refs: 30
ISSN: 1024-5332 E-ISSN: 1607-8454 CODEN: HMATFL
CY United Kingdom
DT Journal; General Review; (Review)
FS 022 Human Genetics
025 Hematology
027 Biophysics, Bioengineering and Medical Instrumentation
LA English
SL English
ED Entered STN: 12 Jan 2006
Last Updated on STN: 12 Jan 2006
AB Artificial intelligence (AI) is a computer based science which aims to simulate human brain faculties using a computational system. A brief

history of this new science goes from the creation of the first artificial neuron in 1943 to the first artificial neural network application to genetic algorithms. The potential for a similar technology in medicine has immediately been identified by scientists and researchers. The possibility to store and process all medical knowledge has made this technology very attractive to assist or even surpass clinicians in reaching a diagnosis. Applications of AI in medicine include devices applied to clinical diagnosis in neurology and cardiopulmonary diseases, as well as the use of expert or knowledge-based systems in routine clinical use for diagnosis, therapeutic management and for ***prognostic*** evaluation. Biological applications include genome sequencing or DNA gene expression microarrays, modeling gene networks, analysis and clustering of gene expression data, pattern recognition in DNA and proteins, protein structure prediction. In the field of hematology the first devices based on AI have been applied to the routine laboratory data management. New tools concern the differential diagnosis in specific diseases such as anemias, thalassemias and leukemias, based on neural networks trained with data from peripheral blood analysis. A revolution in ***cancer*** diagnosis, including the diagnosis of hematological malignancies, has been the introduction of the first microarray based and bioinformatic approach for molecular diagnosis: a systematic approach based on the monitoring of simultaneous expression of thousands of genes using DNA microarray, independently of previous biological knowledge, analysed using AI devices. Using ***gene*** ***profiling*** , the traditional diagnostic pathways move from clinical to molecular based diagnostic systems.

L16 ANSWER 19 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2005246957 EMBASE <<LOGINID::20071005>>
TI ***Gene*** ***profiling*** of high risk neuroblastoma.
AU Vasudevan S.A.; Nuchtern J.G.; Shohet J.M.
CS Dr. J.G. Nuchtern, Pediatric Surgery, Michael E. DeBakey Department of Surgery, Baylor College of Medicine, 6621 Fannin, CC 650.00, Houston, TX 77030, United States. nuchtern@bcm.tmc.edu
SO World Journal of Surgery, (Mar 2005) Vol. 29, No. 3, pp. 317-324.
Refs: 74
ISSN: 0364-2313 E-ISSN: 1432-2323 CODEN: WJSUDI
CY United States
DT Journal; Conference Article; (Conference paper)
FS 016 Cancer
022 Human Genetics
008 Neurology and Neurosurgery
LA English
SL English
ED Entered STN: 30 Jun 2005
Last Updated on STN: 30 Jun 2005
AB Neuroblastoma, a ***cancer*** of young children, is well known for its diverse pattern of presentation. Approximately one-half of children have localized tumors that can be cured with surgery alone. The remaining children have widespread metastatic disease or quite large, aggressive, localized tumors. These children have a poor long-term ***survival*** rate of approximately 30%. We ***review*** the ***prognostically*** significant histologic and molecular features of high risk neuroblastoma and propose an algorithm to dissect further the differentially expressed genes that define the phenotype of this disease. Over the past 25 years, much effort has gone into establishing reliable ***prognostic*** indicators of high risk disease. For neuroblastoma, age, stage, and histopathology have time and again correlated well with outcomes. Chromosomal number, or ploidy, and amplification of the MYCN oncogene have proved to be equally as important and are commonly used to stratify patient risk. Other potentially lucrative markers include chromosome 1p deletion, chromosome 17q gain, receptor tyrosine kinases A and B (trk-A, trk-B), CD44, CXCR4, and multidrug resistance associated protein (MRP). With the onset of new technology, expression microarrays are now being used to profile advanced-stage neuroblastoma on a larger scale. Genes particular to cell cycle control, DNA/RNA replication, ribosomal synthesis, neuronal differentiation, and intracellular/extracellular signal transduction have been identified through differential expression analysis. We present our research on the MYCN transcription factor and target gene, MCM7, to show the utility of this approach. .COPYRGT. 2005 by the Societe Internationale de Chirurgie.

L16 ANSWER 20 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:1268735 CAPLUS <<LOGINID::20071005>>
DN 144:266283
TI Highlights from: Controversies in breast ***cancer*** adjuvant and neoadjuvant therapy
AU Reddy, G. Kesava; O'Shaughnessy, Joyce A.; Nadler, Eric
CS USA
SO Clinical Breast Cancer (2005), 6(4), 288-291
CODEN: CBCLB7; ISSN: 1526-8209
PB Cancer Information Group, LP
DT Journal; General Review
LA English
AB A ***review*** . A discussion on targeting the PI3K/Akt pathway in breast ***cancer*** and the mol. basis of tissue tropism in metastatic breast ***cancer*** is presented. It also discusses the tailoring treatments to subtypes of breast ***cancer*** .
RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 21 OF 43 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN
AN 2005:497575 BIOSIS <<LOGINID::20071005>>
DN PREV200510286687
TI Gene expression profiling in breast ***cancer*** : towards individualising patient management.
AU Murphy, N. [Reprint Author]; Millar, E.; Lee, C. S.
CS Royal Prince Alfred Hosp, Dept Anat Pathol, Missenden Rd, Camperdown, NSW
2050, Australia
n.murphy@garvan.org.au
SO Pathology, (AUG 2005) Vol. 37, No. 4, pp. 271-277.
CODEN: PTLGAX. ISSN: 0031-3025.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 16 Nov 2005
Last Updated on STN: 16 Nov 2005
AB Breast ***cancer*** is a complex and clinically heterogeneous disease. The increase in knowledge of breast ***cancer*** biology has led to a number of clinical advances in the treatment of breast ***cancer*** , most notably the implementation of widespread mammography screening and advances in adjuvant treatment of early-stage disease. In the last 20 years, arrays of potential ***prognostic*** and/or predictive markers of breast ***cancer*** have been analysed. However, relatively few have proven to be clinically useful. To date, the only widely accepted markers for routine use in breast ***cancer*** are the oestrogen receptor (ER), progesterone receptor (PR) and human epidermal growth factor receptor, HER-2 (c-erbB2/ neu). Expression microarray technology and laser capture microdissection have now been employed to further our understanding of the molecular pathogenesis of breast ***cancer*** . Recently reported advances in array technology and RNA amplification methods are having a considerable impact in this field, allowing the analysis of pre-malignant and pre-invasive lesions. A number of studies have identified ***prognostic*** and predictive gene 'signatures', whose prediction of disease outcome and response to treatment is superior to conventional ***prognostic*** indicators. Despite major technological advances, a number of confounding issues remain concerning the potential clinical utility of gene expression profiling, including differences in study design, patient selection, array technology, chemistry, and methods of analysis. It seems likely, however, that following careful 'hypothesis driven' validation studies and clinical trials, expression profiling will be applied in the future to identify patient-specific disease profiles and provide rationale for individualised treatment. This ***review*** focuses on the current use and future potential of microarray profiling in breast ***cancer*** .

L16 ANSWER 22 OF 43 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN DUPLICATE 6
AN 2005:293247 BIOSIS <<LOGINID::20071005>>
DN PREV200510077355
TI Epigenetic changes in virus-associated human cancers.
AU Li, Hsin Pai; Leu, Yu Wei; Chang, Yu San [Reprint Author]
CS Ghang Gung Univ, Grad Inst Basic Med Sci, Taoyuan 333, Taiwan
ysc@mail.cgu.edu.tw
SO Cell Research, (APR 2005) Vol. 15, No. 4, pp. 262-271.
ISSN: 1001-0602.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 4 Aug 2005
Last Updated on STN: 4 Aug 2005
AB Epigenetics of human ***cancer*** becomes an area of emerging research direction due to a growing understanding of specific epigenetic pathways and rapid development of detection technologies. Aberrant promoter hypermethylation is a prevalent phenonmena in human cancers. Tumor suppressor genes are often hypermethylated due to the increased activity or deregulation of DNMTs. Increasing evidence also reveals that viral genes are one of the key players in regulating DNA methylation. In this ***review*** , we will focus on hypermethylation and tumor suppressor gene silencing and the signal pathways that are involved, particularly in cancers closely associated with the hepatitis B virus, simian virus 40 (SV40), and Epstein-Barr virus. In addition, we will discuss current technologies for genome-wide detection of epigenetically regulated targets, which allow for systematic DNA hypermethylation analysis. The study of epigenetic changes should provide a global view of ***gene*** ***profile*** in ***cancer*** , and epigenetic markers could be used for early detection, ***prognosis*** , and therapy of ***cancer*** .

L16 ANSWER 23 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:890002 CAPLUS <<LOGINID::20071005>>
DN 143:226830
TI The role of DNA-microarray in translational ***cancer*** research
AU Korfee, Soenke; Eberhardt, Wilfried; Fujiwara, Yasuhiro; Nishio, Kazuto
CS Shien Lab, National Cancer Center Hospital, Tokyo, 104-0045, Japan
SO Current Pharmacogenomics (2005), 3(3), 201-216
CODEN: CPUHAC; ISSN: 1570-1603
PB Bentham Science Publishers Ltd.
DT Journal; General Review
LA English
AB A ***review*** . The overall ***prognosis*** for the majority of

cancer patients remains poor. Current conventional strategies in clin. ***cancer*** research are unable to adequately answer a large no. of important unsolved questions. Although some patients achieve substantial benefits from classical cytotoxic chemotherapy, others will not. The mechanisms behind this phenomenon are still not identified in detail. Furthermore, the activity of promising novel mol. targeting anticancer agents like tyrosine kinase inhibitors is currently not predictable within the individual patient. The biol. background for this clin. and ***prognostic*** heterogeneity in behavior is more or less the large individual variation in the biol. nature of tumors within the same classified histol. subgroup. The overall usefulness of conventional histopathol. classifications to adequately predict patient ***prognosis*** or response to chemotherapy is limited. The most promising way to solve this issue is to found clin. research strategies on basic biol. evidence. New genomic technologies have been developed within recent years. These techniques are able to analyze thousands of genes and their expression profiles simultaneously. An increasing no. of investigations has reported applications of these novel technologies within clin. trials settings. The aim of this approach is to identify new subsets of ***cancer*** patients, to improve prediction of their clin. outcome or response to treatment and select new targets for innovative therapeutic drugs based on the findings from gene expression profiles. Results of these gene expression profile studies could potentially lead to more individually tailored systemic ***cancer*** therapy. In the recent years, a remarkable no. of studies based on these techniques have already been reported. Although the published results are clearly impressive and highly promising, a lot of work remains to be done. Moreover, there is a strong need for an increase in reliability and reproducibility of such gene expression profiling techniques and thus introduction of reproducible quality control in the performance of these assays. Although a large no. of issues remain to be clarified prior to a more general application of genomic profiling techniques in clin. ***cancer*** research, this strategy will eventually turn out as a promising approach to improve successful management of ***cancer*** patients.

RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 24 OF 43 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 7

AN 2006:239493 BIOSIS <<LOGINID::20071005>>
DN PREV200600241630
TI Genomics of prostate ***cancer*** : Is there anything to "translate"?.
AU Kopper, Laszlo [Reprint Author]; Timar, Jozsef
CS Semmelweis Univ, Inst Pathol and Expt Canc Res 1, Ulloi Ut 78, H-1085 Budapest, Hungary
kopper@korbl.sote.hu
SO Pathology & Oncology Research, (2005) Vol. 11, No. 4, pp. 197-203. ISSN: 1219-4956.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 19 Apr 2006
Last Updated on STN: 19 Apr 2006
AB This ***review*** provides an up-dated collection of data concerning the genetic and epigenetic changes during development, growth and progression of prostate ***cancer*** . Hereditary and susceptibility factors have a long list, similarly to the expression of single genes connected to various cell functions. It was a hope that covering a large set of genes, array technologies would clarify very rapidly the role of genetics in malignant diseases, offering targets for molecular diagnostics and therapy. The power of high-throughput techniques for the detection and global analysis of gene expression is unquestionable, interesting, astonishing as well as puzzling data have already been obtained. However, the standardization of the procedures is still missing and the reproducibility is rather low in many instances. Moreover, the different array methods can select different gene expression profiles, which makes the decision rather difficult. Another important question is, coming again from the array technologies, how far the genotype (the ***gene*** ***profiles*** or fingerprints) can reflect the actual phenotype in a highly complex and readily changing disease as ***cancer*** . Proteomics will provide a closer look to this seemingly unanswerable problem. We are at the beginning of the exploration of the behavior of ***cancer*** cells in order to apply a more effective therapy based on a more reliable set of diagnostic and ***prognostic*** informations.

L16 ANSWER 25 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 8

AN 2005:1110491 CAPLUS <<LOGINID::20071005>>
DN 143:323356
TI Prediction of disease progression and therapy response through molecular markers in breast ***cancer***
AU Hauenstein, Evelyn; Kuschel, Bettina; Meindl, Alfons; Schmitt, Manfred; Kiechle, Marion; Harbeck, Nadia
CS Frauenklinik der Technischen Universitat Munchen, Germany
SO Medizinische Genetik (2005), 17(2), 176-182
CODEN: MGENEZ; ISSN: 0936-5931
PB Verlag Medizinischegenetik
DT Journal; General Review
LA German
AB A ***review*** . Breast ***cancer*** is - like other carcinomas - a disease characterized by certain genetic events in the course of carcinogenesis. It is important to gain a better understanding of the nature and time course of those events. Simultaneously, many efforts were made to avoid either over- or under-therapy and to classify tumors better according to their aggressivity and therapy. For achieving a better classification of tumors, the existing established clin. and histopathol. parameters are not sufficient. Regarding the heterogeneity of tumors, multigen analyses offer a promising prospect to establish such ***gene*** ***profiles*** as new ***prognostic*** and predictive markers and evaluate them for clin. use. For some RNA-based concepts, clin. studies for validation and therapy are already being planned. Also, first clin. data for DNA-based techniques describing epigenetic phenomena were validated. Their correlation with disease progress and therapy response is significant and reproducible. Still before such mol. tests can be used for everyday clin. practice, certain quality criteria have to be fulfilled. Also as long as there is no reliable methodical and clin. validation of the existing data, the use of multigen analyses in breast carcinomas should be limited to clin. studies.

RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 26 OF 43 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 2006:208053 BIOSIS <<LOGINID::20071005>>
DN PREV200600209781
TI Improved ***survival*** in colorectal cancers with adenomas.
AU Mattar, Mark; Frankel, Paul; Clarke, Kevin; Yen, Yuri; David, Donald; Chu, David
SO Gastroenterology, (APR 2005) Vol. 128, No. 4, Suppl. 2, pp. A151. Meeting Info.: Annual Meeting of the American-Gastroenterological-Association/Digestive-Disease-Week. Chicago, IL, USA. May 14 -19, 2005. Amer Gastroenterol Assoc.
CODEN: GASTAB. ISSN: 0016-5085.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 29 Mar 2006
Last Updated on STN: 29 Mar 2006
AB INTRODUCTION: Colorectal cancers (CRC) evolve from a multi-step tumorigenesis and morphologically are characterized by the adenoma. CRC with adenomas (CRC+A) have distinct clinical features, including reports of improved ***survival*** . We hypothesize that this ***survival*** advantage, conferred by the presence of adenomas, is related to inherent biological differences in CRC+A and not due to earlier diagnosis or stage of presentation.METHODS: We conducted a retrospective chart ***review*** of 569 patients treated at City of Hope Medical Center from 1983-2003. Data on age, sex, ***survival*** , CRC stage, location, recurrence and adenoma, number, size, histology, location, and prior colonoscopy history were entered into an Excel database and analyzed.RESULTS: The mean age was 62 years (range 17-90) and 54% were males, The majority of CRC were left sided (67%). The stage distribution was 0/I 12%, 11 21%, 111 34% and stage IV 33%. CRC with synchronous adenomas (+SA) was 33% and CRC+A 42%. The event-free ***survival*** and overall ***survival*** favored CRC+A. After adjusting for age, AJCC Stage, gender and total number of colonoscopic examinations, the relative risk (RR) for an event was 1.51 ($p<0.003$) for patients without adenomas compared to those with adenomas.CONCLUSIONS: CRC+A represent a distinct population of patients with CRC. The apparent association seems to confer a ***survival*** advantage. This difference could not be explained based on age, gender or stage. The ***survival*** benefit, although slightly less dramatic, remained significant even when we controlled for number of repeat colonoscopies. We are planning to investigate whether these clinical differences will translate into biologically distinct entities demonstrating different molecular and ***gene*** ***profiles*** .[GRAPHICS]

L16 ANSWER 27 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN

AN 2005:654721 CAPLUS <<LOGINID::20071005>>
DN 143:150582
TI Biological significance of lowered tissue oxygen pressure in pituitary adenomas: A ***review*** article
AU Yoshida, Daizo; Kim, Kyonsong; Teramoto, Akira
CS Dep. Neurosurg., Nippon Med. Sch. Second Hosp., Kawasaki, 221-0063, Japan
SO Nippon Ika Daigaku Igakkai Zasshi (2005), 1(3), 110-116
CODEN: NIDIB3; ISSN: 1349-8975
PB Nippon Ika Daigaku Igakkai
DT Journal; General Review
LA Japanese
AB A ***review*** . Hypoxia, the disruption of oxygen homeostasis induced by low oxygen supply, is crit. in the development and progression of a large no. of tumors. Various solid tumors are basically in a hypoxic condition, when growth exceeds vascular supply. Under such conditions, cellular oxygen concn. redirects cellular biosynthetic pathways to promote adaptation and enable ***survival*** . Recently, a transcriptional factor called hypoxia-inducible factor (HIF)-1.alpha. has been shown to play a crucial role in the regulation of many genes involved in the hypoxia adaptive pathway, esp. vascular endothelial growth factor (VEGF). Under hypoxic conditions, many tumor cells promote angiogenesis via

HIF-1.alpha.. Meanwhile pituitary tumors are solid tumors in which the regional oxygen satn. is lower than that of normal pituitary gland and the vasculature is usually poor. Despite expression of HIF-1.alpha. was confined in pituitary adenomas, its function driving to angiogenesis, apoptotic induction, and cell invasion, even though these issues have been extended in the other malignant tumors in recent years, has not yet been discussed. We have investigated the expression of microvascular d., HIF-1.alpha., and VEGF in primary human pituitary adenomas focusing on the co-localization, and subsequently in vitro study, elucidated ***gene*** ***profiling*** regulated by HIF-1.alpha.. Our previous studies indicated that HIF-1.alpha. immunoreactivity was confined to the nucleoplasm, but was present in both tumor cells and vascular endothelial cells. There was no difference in microvascular d. by histotype. ACTH-producing adenomas showed the lowest level of HIF-1.alpha., whereas PRL-producing adenomas and HIF-1.alpha.-pos. microvessels showed the highest ($p < 0.001$). There was no significant correlation in the expression levels of HIF-1.alpha. mRNA and VEGF mRNA in pituitary adenomas. Both of HIF-1.alpha. and VEGF proteins expressed in pituitary adenoma and they were, in part, co-localized. Transfection with specific siRNA duplexes knocked down HIF1-.alpha. mRNA and protein expression in hypoxia-exposed cells by approx. 80%. Microarray anal. indicated that HIF1-.alpha. down-regulated caspase-10, but up-regulated of laminin .beta.2 (4.26 folds), SAP90 (3.34 folds), and BNIP3 (3.24 folds). Conclusively in these poorly vascularized tumors, HIF-1.alpha. may not mainly regulate the VEGF expression in pituitary adenoma. In vitro studies strongly suggest that HIF 1-.alpha. exerts an antiapoptotic role in HP-75 in hypoxia mediated by down-regulation of caspase-10 and that hypoxia can potentially enhance the cell invasion properties of a pituitary adenoma cell line through elevated expression of laminin .beta.-2. The mechanism of tumor angiogenesis and cell invasion in pituitary adenomas may differ from that in the other ***cancer*** cells.

L16 ANSWER 28 OF 43 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2005:284556 BIOSIS <<LOGINID::20071005>>
DN PREV200510066648
TI Gene expression arrays in ***cancer*** research: methods and applications.
AU Brentani, Ricardo R. [Reprint Author]; Carraro, Dirce Maria; Verjovski-Almeida, Sergio; Reis, Eduardo M.; Neves, E. Jordao; de Souza, Sandro J.; Carvalho, Alex F.; Brentani, Helena; Reis, Luiz F. L.
CS Rua Prof Antonio Prudente 109,4th Floor, BR-01509010 Sao Paulo, Brazil rbrentani@ludwig.org.br
SO Critical Reviews in Oncology-Hematology, (MAY 2005) Vol. 54, No. 2, pp. 95-105.
ISSN: 1040-8428.
DT Article
LA English
ED Entered STN: 27 Jul 2005
Last Updated on STN: 27 Jul 2005
AB During the last 5 years, the number of papers describing data obtained by microarray technology increased exponentially with about 3000 papers in 2003. Undoubtedly, ***cancer*** is by far the disease that received most of the attention as far as the amount of data generated. As array technology is rather new and highly dependent on bioinformatics, mathematics and statistics, a clear understanding of the knowledge and information derived from array-based experiments is not widely appreciated. We shall ***review*** herein some of the issues related to the construction of DNA arrays, quantities and heterogeneity of probes and targets, the consequences of the physical characteristics of the probes, data extraction and data analysis as well as the applications of array technology.Our goal is to bring to the general audience, some of the basics of array technology and its possible application in oncology. By discussing some of the basic aspects of the methodology, we hope to stimulate criticism concerning the conclusions proposed by authors, especially in the light of the very low degree of reproducibility already proven when commercially available platforms were compared [1]. Regardless of its pitfalls, it is unquestionable that array technology will have a great impact in the management of ***cancer*** and its applications will range from the discovery of new drug targets, new molecular tools for diagnosis and ***prognosis*** as well as for a tailored treatment that will take into account the molecular determinants of a given tumor. Hence, we shall also highlight some of the already available and promising applications of array technology on the day-to-day practiceof oncology. (c) 2005 Elsevier Ireland Ltd. All rights reserved.

L16 ANSWER 29 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2005192076 EMBASE <<LOGINID::20071005>>
TI ***Gene*** ***profiling*** in squamous cell carcinoma of the head and neck.
AU Akervall J.
CS Dept. Otolaryngol., Hd. Neck Surg., University Hospital, Sweden. akervall@rontalclinic.com
SO Cancer and Metastasis Reviews, (Jan 2005) Vol. 24, No. 1, pp. 87-94.
Refs: 29
ISSN: 0167-7659 CODEN: CMRED4
CY Netherlands
DT Journal; General Review; (Review)
FS 016 Cancer
022 Human Genetics
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
LA English
SL English
ED Entered STN: 19 May 2005
Last Updated on STN: 19 May 2005
AB ***Survival*** for patients with squamous cell carcinoma of the head and neck (SCCHN) is still poor, despite great technical improvements in radiotherapy and surgery. A possible explanation for this is the lack of individualization in treatment based on biological properties of the tumors, resulting in over- as well as under treatment. Management of SCCHN has mainly been based on TNM classification over the last decades. However, a large amount of studies have shown that biomarkers may add ***prognostic*** information, independently of the TNM system, indicating that biological aggressiveness is not entirely reflected by the T- and N-status of the tumor. A conclusion to draw from this is that the present standardized treatment based on macroscopic features of the tumor in many cases will result in suboptimal treatment since important underlaying genetic properties of the tumors are not taken into consideration. A variety of laboratory techniques have been used in studies that investigate the individual biological features, spanning from methods that screen the genome for chromosomal and genetic abnormalities, e.g. cytogenetics, CGH, SKY, cDNA micro array to detailed studies of specific aberrations, e.g. southern, northern and western blotting, PCR based analysis and immunohistochemistry. Dysregulation of genes involved in e.g. cell cycle control, proliferation, drug resistence, and metastasis have been linked to outcome of treatment and ***survival*** . The purpose of this ***review*** of the literature was to summarize what has been studied so far by cDNA micro array techniques with regards to genetic screening in general and biomarkers that relate to response to therapy and prediction of clinical outcome in particular. We conclude that the majority of investigations that focus on ***gene*** ***profiling*** have a descriptive character, e.g. comparisons of tumor and normal cells, metastatic and non-metastatic properties, and differences between sub-sites and grades of differentiation. There are just a handful studies that so far have investigated how ***gene*** ***profiling*** can be used to predict clinical course. .COPYRGT. 2005 Springer Science + Business Media, Inc.

L16 ANSWER 30 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:351455 CAPLUS <<LOGINID::20071005>>
DN 144:429966
TI ***Prognostic*** significance of cyclin E in breast ***cancer***
AU Potemski, Piotr; Kordek, Radzislaw
CS Klinika Chemioterapii Nowotworow, Katedra Onkol., Wojewodzki Szpital Specjalistyczny im. Mikolaja Kopernika, Uniw. Med., Lodz, 93-509, Pol.
SO Onkologia w Praktyce Klinicznej (2005), 1(2), 76-82
CODEN: OPKNBB; ISSN: 1734-3542
PB Wydawnictwo Via Medica
DT Journal; General Review
LA Polish
AB A ***review*** . In the present ***review*** the role of cyclin E and other cell cycle mols. as ***prognostic*** markers in breast ***cancer*** is discussed Cell cycle transitions are controlled by cyclin-dependent protein kinases. Cyclin E is an important regulator of cell cycle progression from phase G1 to synthetic phase. The highest concn. of cyclin E in the cell is obsd. near restriction point in the late G1 phase. In breast ***cancer*** the aberrant expression of cyclin E has been frequently described. In breast ***cancer*** cells full-length cyclin E is degradated by proteolysis to its low mol. wt. isoforms. Overexpression of both cyclin E and low mol. wt. isoforms gives tumor cells growth advantage. Expression of cyclin E and other cell cycle mols. can be evaluated by immunohistochem., tissue microarrays, various blot assays, reverse transcription-polymerase chain reaction and DNA microarrays. The results of numerous studies show that overexpression of cyclin E may be an important ***prognostic*** factor. However, in some models of multivariate anal. this effect was eliminated by estrogen receptors presence or other classical ***prognostic*** factors. However, futher confirmatory studies are warranted as overexpression of cyclin E may also be just a part of ***gene*** ***profile*** and not a single, independent ***prognostic*** factor.

L16 ANSWER 31 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:1321256 CAPLUS <<LOGINID::20071005>>
DN 144:304295
TI Gene-expression profiling and the future of adjuvant therapy
AU Van De Vijver, Marc
CS Department of Pathology, Netherlands Cancer Institute, Amsterdam, Neth.
SO Oncologist (2005), 10(Suppl. 2), 30-34
CODEN: OCOLF6; ISSN: 1083-7159
PB AlphaMed Press
DT Journal; General Review
LA English
AB A ***review*** . Gene-expression profiling can help distinguish between patients at high risk and those at low risk for developing distant metastases, and so identify patients for adjuvant therapy. For several years, the Netherlands ***Cancer*** Institute has been working on gene-expression profiling of breast ***cancer*** using a microarray platform contg. 25,000 genes. Using supervised classification, a ***prognostic*** classifier consisting of 70 genes could be identified. In addn. to providing ***prognostic*** information, ***gene*** ***profiling*** should also enable us to detect patients who are likely

to respond to particular adjuvant interventions. Well-known predictors for response to systemic therapy include estrogen receptor status HER-2 status, c-kit mutation, and epidermal growth factor receptor mutation. Because of the long periods required for predicting responsiveness in the adjuvant setting, neoadjuvant trials promise far quicker results. Several neoadjuvant studies are under way or planned to investigate gene-expression profiling as a means of predicting the therapeutic response to docetaxel (Taxotere; Aventis Pharmaceuticals Inc., Bridgewater, NJ, http://www.aventispharma-us.com), paclitaxel (Taxol; Bristol-Myers Squibb, Princeton, NJ, http://www.bms.com), cyclophosphamide, and doxorubicin (Adriamycin; Bedford Labs., Bedford, OH, http://www.bedfordlabs.com) in breast ***cancer*** patients. It is expected that in the coming years an increasing no. of novel ***prognostic*** and predictive tests will help in guiding the adjuvant systemic treatment of breast ***cancer*** and other malignancies.
RE.CNT 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 32 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN DUPLICATE 9
AN 2006579944 EMBASE <<LOGINID::20071005>>
TI ***Prognosis*** of follicular lymphomas.
AU Solal-Celigny P.
CS P. Solal-Celigny, Department of Hematology and Medical Oncology, Centre Jean Bernard, 9 Rue Beauverger, 72000 Le Mans, France. p.solal-celigny@noos.fr
SO Clinical Lymphoma and Myeloma, (Jun 2005) Vol. 6, No. 1, pp. 21-25.
Refs: 32
ISSN: 1557-9190
CY United States
DT Journal; General Review; (Review)
FS 016 Cancer
025 Hematology
029 Clinical and Experimental Biochemistry
LA English
SL English
ED Entered STN: 19 Dec 2006
Last Updated on STN: 19 Dec 2006
AB Although numerous treatment approaches are proposed for patients with follicular lymphoma, criteria to help in choosing a treatment for a given patient and for comparing trial results are lacking. Several retrospective studies have analyzed ***prognostic*** factors, but their conclusions rely on limited numbers of patients treated during long periods, and their results are discordant. The Follicular Lymphoma International ***Prognostic*** Index was designed from the data recorded over 8 years of nearly 5000 patients registered worldwide. Five factors are used (age, Ann Arbor stage, number of nodal sites, serum lactate dehydrogenase level, and hemoglobin level) to build a 3-category index. This index, together with new biologic markers such as ***gene*** ***profiling*** and proteomics, could help provide an optimal treatment option for patients with follicular lymphoma.

L16 ANSWER 33 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2005012452 EMBASE <<LOGINID::20071005>>
TI DNA array-based ***gene*** ***profiling*** : From surgical specimen to the molecular portrait of ***cancer*** .
AU Mocellin S.; Provenzano M.; Rossi C.R.; Pilati P.; Nitti D.; Lise M.
CS Dr. S. Mocellin, Clinica Chirurgica Generale II, Dipto. di Sci. Oncol. e Chirurgiche, Universita di Padova, Via Giustiniani, 2, 35128 Padova, Italy. mocellins@hotmail.com
SO Annals of Surgery, (Jan 2005) Vol. 241, No. 1, pp. 16-26.
Refs: 116
ISSN: 0003-4932 CODEN: ANSUA5
CY United States
DT Journal; General Review; (Review)
FS 016 Cancer
022 Human Genetics
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
009 Surgery
LA English
SL English
ED Entered STN: 20 Jan 2005
Last Updated on STN: 20 Jan 2005
AB ***Cancer*** is a heterogeneous disease in most respects, including its cellularity, different genetic alterations, and diverse clinical behaviors. Traditional molecular analyses are reductionist, assessing only 1 or a few genes at a time, thus working with a biologic model too specific and limited to confront a process whose clinical outcome is likely to be governed by the combined influence of many genes. The potential of functional genomics is enormous, because for each experiment, thousands of relevant observations can be made simultaneously. Accordingly, DNA array, like other high-throughput technologies, might catalyze and ultimately accelerate the development of knowledge in tumor cell biology. Although in its infancy, the implementation of DNA array technology in ***cancer*** research has already provided investigators with novel data and intriguing new hypotheses on the molecular cascade leading to carcinogenesis, tumor aggressiveness, and sensitivity to antiblastic agents. Given the revolutionary implications that the use of this technology might have in the clinical management of patients with ***cancer*** , principles of DNA array-based tumor ***gene*** ***profiling*** need to be clearly understood for the data to be correctly interpreted and appreciated. In the present work, we discuss the technical features characterizing this powerful laboratory tool and ***review*** the applications so far described in the field of oncology.

L16 ANSWER 34 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2005523442 EMBASE <<LOGINID::20071005>>
TI RB1 gene mutation up-date, a meta-analysis based on 932 reported mutations available in a searchable database.
AU Valverde J.R.; Alonso J.; Palacios I.; Pestana A.
CS A. Pestana, Oncolab, Departamento de Biologia Molecular y Celular del Cancer, Instituto de Investigaciones Biomedicas A. Sols, 28029 Madrid, Spain. apestana@iib.uam.es
SO BMC Genetics, (4 Nov 2005) Vol. 6. arn. 53.
Refs: 47
ISSN: 1471-2156 E-ISSN: 1471-2156 CODEN: BGMEDS
CY United Kingdom
DT Journal; Article
FS 012 Ophthalmology
016 Cancer
022 Human Genetics
027 Biophysics, Bioengineering and Medical Instrumentation
LA English
SL English
ED Entered STN: 15 Dec 2005
Last Updated on STN: 15 Dec 2005
AB Background. Retinoblastoma, a prototype of hereditary ***cancer*** , is the most common intraocular tumour in children and potential cause of blindness from therapeutic eye ablation, second tumours in germ line carrier's ***survivors*** , and even death when left untreated. The molecular scanning of RB1 in search of germ line mutations lead to the publication of more than 900 mutations whose knowledge is important for genetic counselling and the characterization of phenotypic-genotypic relationships. Results. A searchable database (RBGMdb) has been constructed with 932 published RB1 mutations. The spectrum of these mutations has been analyzed with the following results: 1) the retinoblastoma protein is frequently inactivated by deletions and nonsense mutations while missense mutations are the main inactivating event in most genetic diseases. 2) Near 40% of RB1 gene mutations are recurrent and gather in sixteen hot points, including twelve nonsense, two missense and three splicing mutations. The remainder mutations are scattered along RB1, being most frequent in exons 9, 10, 14, 17, 18, 20, and 23. 3) The analysis of RB1 mutations by country of origin of the patients identifies two groups in which the incidence of nonsense and splicing mutations show differences extremely significant, and suggest the involvement of predisposing ethnic backgrounds. 4) A significant association between late age at diagnosis and splicing mutations in bilateral retinoblastoma patients suggests the occurrence of a delayed-onset genotype. 5) Most of the reported mutations in low-penetrance families fall in three groups: a) Mutations in regulatory sequences at the promoter resulting in low expression of a normal Rb; b) Missense and in-frame deletions affecting non-essential sequence motifs which result in a partial inactivation of Rb functions; c) Splicing mutations leading to the reduction of normal mRNA splicing or to alternative splicing involving either true oncogenic or defective (weak) alleles. Conclusions. The analysis of RB1 gene mutations logged in the RBGMdb has shown relevant phenotype-genotype relationships and provided working hypothesis to ascertain mechanisms linking certain mutations to ethnicity, delayed onset of the disease and low-penetrance. ***Gene*** ***profiling*** of tumors will help to clarify the genetic background linked to ethnicity and variable expressivity or delayed onset phenotypes. .COPYRGT. 2005 Valverde et al., licensee BioMed Central Ltd.

L16 ANSWER 35 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:650414 CAPLUS <<LOGINID::20071005>>
DN 142:277389
TI Biomarkers in breast ***cancer***
AU Kurebayashi, Junichi
CS Dep. Breast and Thyroid Surgery, Kawasaki Med. Sch., Kurashiki, Okayama, 701-0192, Japan
SO Gan to Kagaku Ryoho (2004), 31(7), 1021-1026
CODEN: GTKRDX; ISSN: 0385-0684
PB Gan to Kagaku Ryohosha
DT Journal; General Review
LA Japanese
AB A ***review*** . Biomarkers are measured in the management of breast ***cancer*** patients for the following purposes. (1) Early detection of breast ***cancer*** : blood tumor markers such as CA 15-3 are useless for this detection because of a low sensitivity. Proteomics profiling has recently been investigated using blood or nipple aspirate fluid for the detection. Measurement of CEA and HER 2 in abnormal nipple discharge has been approved for diagnosis of breast ***cancer*** in Japan. (2) Monitoring of breast ***cancer*** patients: serum tumor markers are routinely measured for early detection of recurrent diseases, evaluation of therapeutic response and monitoring outcome of patients by a majority of breast ***cancer*** experts in Japan. Study results investigated by the Study Group of the Japanese Breast ***Cancer*** Society in 2001 are presented with regard to the questionnaire survey on the present status of tumor marker measurement and the clin. study on usefulness of tumor markers for the evaluation for therapeutic response. (3) ***Prognostic*** factors: new biomarkers have been investigated to select patients at high risk for distant metastases, which could not be

selected by classic ***prognostic*** factors. Three ***prognostic*** factors (UPA/PAI-1, cyclin E, ***gene*** ***profiling***), which were discussed at the 8th St. Gallen International Consensus Meeting last year, are mainly discussed. (4) Predictive factors for therapeutic response: hormone receptors (HR) have been used as reliable predictive factors for response to endocrine therapy. Other biomarkers have been investigated to select patients with tumors HR-pos. but unresponsive to endocrine therapy. Current status, clin. significance, problems and future directions on predictive factors for response to cytotoxic chemotherapy are also discussed.

L16 ANSWER 36 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2004207361 EMBASE <<LOGINID::20071005>>
TI Integration of multimodality approaches in the management of malignant pleural mesothelioma.
AU Martino D.; Pass H.I.
CS Dr. H.I. Pass, Harper University Hospital, 3990 John R, Detroit, MI 48201, United States. hpass@dmc.org
SO Clinical Lung Cancer, (Mar 2004) Vol. 5, No. 5, pp. 290-298.
Refs: 95
ISSN: 1525-7304 CODEN: CLCLCA
CY United States
DT Journal; General Review; (Review)
FS 014 Radiology
015 Chest Diseases, Thoracic Surgery and Tuberculosis
016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
039 Pharmacy
LA English
SL English
ED Entered STN: 4 Jun 2004
Last Updated on STN: 4 Jun 2004
AB More than half a century after the first descriptions of mesothelioma as a pathologic entity, satisfactory treatment is still elusive. Although relatively uncommon, the incidence of mesothelioma will most likely increase over the next 10-20 years. Advances have been made in understanding the pathogenesis, diagnosis, and staging, but they have not translated into markedly improved ***survival*** . Some use palliative treatment as the primary means of therapy even now. On the other hand, a cadre of individuals have studied how surgery, chemotherapy, and radiation therapy affect the disease. Although each individual modality has had limited success by itself, a multimodality approach has been documented to improve ***survival*** and quality of life. In addition, intriguing discoveries in immunology and ***gene*** ***profiling*** and therapy promise hope for further improvement. In this article, we will illustrate the current views on integrating these different approaches and delineate areas of active research.

L16 ANSWER 37 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2005082809 EMBASE <<LOGINID::20071005>>
TI Predicting the outcome of breast ***cancer*** treatment by ***gene*** ***profiling*** .
AU Stenger M.; Schwartzberg L.S.
CS M. Stenger, West Clinic, Memphis, TN, United States
SO Community Oncology, (Sep 2004) Vol. 1, No. 3, pp. 138+140.
Refs: 4
ISSN: 1548-5315
CY United States
DT Journal; General Review; (Review)
FS 016 Cancer
022 Human Genetics
027 Biophysics, Bioengineering and Medical Instrumentation
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
LA English
SL English
ED Entered STN: 3 Mar 2005
Last Updated on STN: 3 Mar 2005
AB Correlating pretreatment gene expression with clinical outcome is proving efficacious in breast tumors. The techniques are broadening the possibilities for rational treatment of breast ***cancer*** patients.

L16 ANSWER 38 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:778411 CAPLUS <<LOGINID::20071005>>
DN 139:346410
TI Extending the utility of ***gene*** ***profiling*** data by bridging microarray platforms
AU Ferl, Gregory Z.; Timmerman, John M.; Witte, Owen N.
CS Biocybernetics Laboratory, University of California, Los Angeles, CA, 90095, USA
SO Proceedings of the National Academy of Sciences of the United States of America (2003), 100(19), 10585-10587
CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal; General Review
LA English
AB A ***review*** . In a recent issue of PNAS, Wright et al. proposed a statistical model that can be used to translate exptl. results across microarray platforms. The model is based on a linear predictor score (LPS) applied to hierarchical clustering results. The model was used to reanalyze oligonucleotide microarray data from a previous study of diffuse large B cell lymphoma tumors, and sep. the tumor samples into three groups corresponding to distinct clin. outcomes. Cross-validation of gene expression results among data sets generated in particular types of ***cancer*** by using methods such as those described by Wright et al. should help to define the genes most relevant for disease classification, ***prognostics*** , and therapeutic targeting.
RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 39 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003235965 EMBASE <<LOGINID::20071005>>
TI [Perspectives for molecular diagnostics exemplified by urothelial bladder carcinoma].
PERSPEKTIVEN DER MOLEKULAREN DIAGNOSTIK DARGESTELLT AM BEISPIEL DES HARNBLASENKARZINOMS.
AU Grimm M.-O.; Burchardt M.; Schulz W.A.
CS M.-O. Grimm, Urologische Klinik, Heinrich-Heine-Universitat, Moorenstrasse 5, 40225 Dusseldorf, Germany. marc-oliver.grimm@uni-duesseldorf.de
SO Urologe - Ausgabe A, (1 May 2003) Vol. 42, No. 5, pp. 650-659.
Refs: 60
ISSN: 0340-2592 CODEN: URGABW
CY Germany
DT Journal; General Review; (Review)
FS 016 Cancer
022 Human Genetics
028 Urology and Nephrology
037 Drug Literature Index
006 Internal Medicine
LA German
SL English; German
ED Entered STN: 26 Jun 2003
Last Updated on STN: 26 Jun 2003
AB The rapidly growing knowledge of molecular mechanisms will change the daily routine of clinicians in the near future. Regarding urothelial bladder carcinoma, one may expect that molecular diagnostics will identify patients susceptible to disease development by screening their genotype. Furthermore, in addition to histopathologic findings, ***prognostic*** markers will be used for disease management. In an ongoing multicenter trial, the decision on whether or not to treat patients with adjuvant chemotherapy after cystectomy is based on their p53 status. In the near future, cytostatic medications are expected to be chosen according to genetic profiles of the tumor or patient. New medications, which target tumor-specific alterations of cell-signaling cascades in bladder or other cancers, prominently inhibitors of the ERBB membrane receptor family, are currently under clinical investigation and will undoubtedly form an important part of therapeutic oncologic regimens. In conclusion, evaluation of ***gene*** ***profiles*** of tumors and patients will gain importance for clinicians.

L16 ANSWER 40 OF 43 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:825336 CAPLUS <<LOGINID::20071005>>
DN 139:393954
TI Breast ***cancer*** gene expression analysis: the case for dynamic profiling
AU Ellis, Matthew J.
CS Duke University Breast Cancer Program, Duke University Medical Center, Durham, NC, 27710, USA
SO Advances in Experimental Medicine and Biology (2003), 532(New Trends in Cancer for the 21st Century), 223-234
CODEN: AEMBAP; ISSN: 0065-2598
PB Kluwer Academic/Plenum Publishers
DT Journal; General Review
LA English
AB A ***review*** . ***Cancer*** is a complicated disease with each individual tumor exhibiting to a variable degree a set of overlapping phenotypes that include abnormal proliferation and cell ***survival*** , genetic instability, tissue invasion, metastasis and angiogenesis. Each of these pathophysiol. functions requires abnormal signaling that fails to respect the normal restraints imposed on healthy cells through homeostatic regulation. Understanding of deregulated gene expression is therefore an essential goal for ***cancer*** research because unraveling this problem will provide insights into the fundamental nature of ***cancer*** as well as provide opportunities for therapeutic intervention. Gene expression profiling is a remarkable technique that addresses this complexity by documenting the expression of thousands of genes at the level of mRNA abundance. From the standpoint of clin. investigation, the initial focus of ***gene*** ***profiling*** expts. was on screening primary tumors for markers of poor ***prognosis*** . However this is only a first step in demonstrating the utility of this technique. Future advances will focus on understanding the evolution of an organ confined primary ***cancer*** to a treatment resistant lethal systemic disease through repeated tumor sampling and anal., an approach termed in this paper "dynamic profiling". Using breast ***cancer*** endocrine therapy as an example our initial approaches to dynamic profiling will be described.
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L16 ANSWER 41 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003110299 EMBASE <<LOGINID::20071005>>
TI The unknown biology of the unknown primary tumour: A literature ***review*** .
AU van de Wouw A.J.; Jansen R.L.H.; Speel E.J.M.; Hillen H.F.P.
CS Dr. A.J. van de Wouw, Department of Internal Medicine, Slingeland Hospital Doetinchem, PO Box 169, 7000 AD Doetinchem, Netherlands. y.van.de.wouw@slingeland.nl
SO Annals of Oncology, (1 Feb 2003) Vol. 14, No. 2, pp. 191-196.
Refs: 75
ISSN: 0923-7534 CODEN: ANONE2
CY United Kingdom
DT Journal; General Review; (Review)
FS 016 Cancer
022 Human Genetics
029 Clinical and Experimental Biochemistry
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
LA English
SL English
ED Entered STN: 27 Mar 2003
Last Updated on STN: 27 Mar 2003
AB The unknown primary tumour (UPT) is an intriguing clinical phenomenon found in approximately 5% of all newly diagnosed patients with ***cancer*** . It is unclear whether UPT forms a distinct biological entity with specific genetic and phenotypic characteristics, or whether it is merely a clinical presentation of metastases in patients in whom the primary tumour cannot be detected and does not result in any visible clinical signs. Understanding the basic biology of UPT may shed light on this issue and, moreover, may have a direct impact on clinical care. A ***review*** of the literature revealed only a limited number of publications describing the genetic and phenotypic features of UPT, most of which focus only on the potential of these markers to predict ***prognosis*** . The question as to whether the biology of UPT is different from tumours of known primaries therefore remains unanswered. Further insight into the molecular mechanisms underlying the oncogenesis of UPT, e.g. by applying newly available DNA and ***gene*** ***profiling*** microarray techniques, will be necessary to understand its specific biology and to develop more effective treatments.

L16 ANSWER 42 OF 43 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2003003989 EMBASE <<LOGINID::20071005>>
TI Minimising the long-term adverse effects of childhood leukaemia therapy.
AU Langebrake C.; Reinhardt D.; Ritter J.
CS Dr. C. Langebrake, Univ. Childrens Hospital Munster, Department of Paediatric Haematology, Albert-Schweitzer-Strasse 33, 48149 Munster, Germany. langebra@uni-muenster.de
SO Drug Safety, (2002) Vol. 25, No. 15, pp. 1057-1077.
Refs: 172
ISSN: 0114-5916 CODEN: DRSAEA
CY New Zealand
DT Journal; General Review; (Review)
FS 016 Cancer
037 Drug Literature Index
038 Adverse Reactions Titles
039 Pharmacy
007 Pediatrics and Pediatric Surgery
LA English
SL English
ED Entered STN: 9 Jan 2003
Last Updated on STN: 9 Jan 2003
AB Malignancies in childhood occur with an incidence of 13-14 per 100 000 children under the age of 15 years. Acute lymphoblastic leukaemia with an incidence of 29% is the most common paediatric malignancy, whereas acute myeloid leukaemias account for about 5%. The treatment of acute leukaemias consists of sequential therapy cycles (induction, consolidation, intensification, maintenance therapy) with different cytostatic drugs over a time period of up to 1.5-3 years. Over the last 25 years of clinical trials, a significant rise in the rate of complete remissions as well as an increase in long-term ***survival*** has been achieved. Therefore, growing attention is now focused on the long-term effects of anti-leukaemic treatment. Several cytostatic drugs administered in the treatment of acute leukaemia in childhood are known to cause long-term adverse effects. Anthracyclines may induce chronic cardiotoxicity, alkylating agents are likely to cause gonadal damage and secondary malignancies and the use of glucocorticoids may cause osteonecrosis. Most of the long-term adverse effects have not been analysed systematically. Approaches to minimising long-term adverse effects without jeopardising outcome have included: (i) the design of new drugs such as a liposomal formulation of anthracyclines, the development of anthracycline-derivates with lower toxicity, the development of cardioprotective agents or, more recently, the use of targeted therapy; (ii) alternative administration schedules like continuous infusion or timed sequential therapy; and (iii) risk group stratification by the monitoring of minimal residual disease. Several attempts have been made to minimise the cardiotoxicity of anthracyclines: decreasing concentrations delivered to the myocardium by either prolonging infusion time or using liposomal formulated anthracyclines or less cardiotoxic analogues, or the additional administration of cardioprotective agents. The advantage of these approaches is still controversial, but there are ongoing clinical trials to evaluate the long-term effects. The use of new diagnostic methods, such as diagnosis of minimal residual disease, which allow reduction or optimisation of dose, offer potential advantages compared with conventional treatment in terms of reducing the risk of severe long-term adverse effects. Most options for minimising long-term adverse effects have resulted from theoretical models and in vitro studies, but only some of the modalities such as the use of dexrazoxane, the continuous infusion of anthracyclines or timed sequential therapy, have been evaluated in prospective, randomised studies in patients. Future approaches to predict severe toxicity may be based upon pharmacogenetics and ***gene*** ***profiling*** .

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AN 2001092515 EMBASE <<LOGINID::20071005>>
TI DNA microarrays in pediatric ***cancer*** .
AU Triche T.J.; Schofield D.; Buckley J.
CS Dr. T.J. Triche, Children's Hospital, MS43, 4650 Sunset Boulevard, Los Angeles, CA 90027, United States
SO Cancer Journal, (2001) Vol. 7, No. 1, pp. 2-15.
Refs: 68
ISSN: 1528-9117 CODEN: CAJOCB
CY United States
DT Journal; General Review; (Review)
FS 016 Cancer
007 Pediatrics and Pediatric Surgery
LA English
SL English
ED Entered STN: 29 Mar 2001
Last Updated on STN: 29 Mar 2001
AB Childhood ***cancer*** , like all ***cancer*** , is at heart a genetic disease. Consequently, fundamental understanding of the oncogenic process is likely to be beneficially addressed by genetic methodology. Current methods have largely focused on single-gene defects, like chimeric genes, which are present in many sarcomas and leukemias. Real understanding is more likely to derive from a genome-wide analysis of these malignancies. Recent technologic advances have made it possible to simultaneously assess the entire expressed ***gene*** ***profile*** , or transcriptome, of a given ***cancer*** . Foremost among these methods is gene expression profiling using DNA microarrays. Two basic approaches predominate: spotted arrays and photolithography arrays. Regardless of the method, the resulting information can be used to create disease profiles, but only if appropriate bioinformatic solutions are employed. Common analytic approaches include two-way expression comparisons, or scatter analyses; outlier gene analysis, to identify significantly dysregulated genes: dendrogram analyses, as pioneered by Eisen; cluster analyses to identify diagnostic or biologic groups; and various forms of functional analyses to identify relevant genes and biologic pathways. Studies of both adult and pediatric ***cancer*** have demonstrated the feasibility of such analyses to identify both diagnostic and ***prognostic*** groups of tumors. Acute childhood leukemias have been grouped into myelogenous and lymphoid, and even B- and
T-cell subsets. Breast ***cancer*** ***prognostic*** groups have been identified on the basis of a small subset of expressed genes. In addition, preliminary data on childhood sarcomas appear to identify both diagnostic and ***prognostic*** subsets. Specifically, embryonal rhabdomyosarcoma could be distinguished from alveolar rhabdomyosarcoma, and even morphologically mixed embryonal and alveolar rhabdomyosarcoma showed similar gene expression profiles in both histologies. Further, collaborative studies using clustering analyses appear to identify ***prognostic*** groups of diverse sarcomas. Larger institutional and cooperative group studies are currently underway to validate these preliminary findings.

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